Med
A

(17)

I

Annals of Otology, Rhinology and Laryngology

FOUNDED BY JAMES PLEASANT PARKER.

VOL. XXII, 1913.

EDITORIAL STAFF.

H. W. LOEB, M. D. .... St. Louis.
EDITOR-IN-CHIEF.

COLLABORATORS:

T. MELVILLE HARDIE, M. D.	Chicago.
JAMES T. CAMPBELL, M. D.	Chicago.
G. L. RICHARDS, M. D.	Fall River.
THOS. J. HARRIS, M. D.	New York.
J. L. GOODALE, M. D.	Boston.
SAMUEL E. ALLEN, M. D.	Cincinnati.
CLEMENT F. THEISEN, M. D.	Albany.
GEO. B. WOOD, M. D.	Philadelphia.
G. ALEXANDER, M. D.	Vienna.
WYATT WINGRAVE, M. D.	London.
A. JANSEN, M. D.	Berlin.
JOHN SENDZIAK, M. D.	Warsaw.
W. POSTHUMUS MEYJES, M. D.	Amsterdam.
N. RH. BLEGVAD, M D.	Copenhagen.
HENRY HORN, M. D.	San Francisco.
VICTOR F. LUCCHETTI	San Francisco.

370608
25.8.39

PUBLISHED QUARTERLY

BY JONES H. PARKER,
MERMOD-JACCARD BUILDING,
ST. LOUIS, MO.
1913.

RF
1
A64
v.22

TABLE OF CONTENTS.

I.—Upon the Present Status of Otosclerosis. By Prof. Dr. Alfred Denker, Halle, A./S. 1

II.—The Report of an Examination of Both Temporal Bones From a Hundred and Twenty Individuals in Reference to the Question of Symmetry in Health and Disease. By Arthur Cheatle, M. D., London 19

III.—Report of a Case of Bronchoscopy for Multiple Foreign Bodies (Almond Shell and Pulp) in a Child Two Years of Age, With Some Observations Upon Bronchoscopy in Infants and Young Children. By J. R. Winslow, B. A., M. D., Baltimore 125

IV.—Tumors of the Larynx. By Robert Levy, M. D., Denver 133

V.—Acute Suppuration of the Middle Ear—Its Neglect and Proper Treatment. By Prof. Dr. Gustav Alexander, Vienna 159

VI.—Pneumococcus Infections of the Nose and Throat. By Clement F. Theisen, M. D., Albany 175

VII.—Two Cases of Air Embolus Following Exploratory Puncture of the Antrum of Highmore. By H. M. Bowen, M. D., Toronto 180

VIII.—Nasal Hemorrhage Following Turbinectomy in a Hemophiliac Treated by the Injection of Human Blood Serum. By Linn Emerson, M. D., Orange 183

IX.—On the Function of the Tonsils. By Louis M. Freedman, M. D., Denver 186

X.—Submucous Resection of the Nasal Septum in the Semirecumbent Position. By William R. Butt, M. D., Philadelphia 201

XI.—Treatment of Deafness by Reeducating the Hearing. By Dr. Maurice, Paris 211

XII.—Removal of Adenoids by Direct Inspection. By Joseph C. Beck, M. D., Chicago........273

XIII.—When to Operate on the Labyrinth in Labyrinth Infection Secondary to Purulent Otitis Media. By G. E. Shambaugh, M. D., Chicago........303

XIV.—Some Attempts at the Intranasal Transplantation of Nasal Tissues. By Samuel Iglauer, B. S., M. D., Cincinnati........308

XV.—The Report of a Case of Paracoustic Vertigo and Nystagmus Cured by Operation on the Labyrinth. By John Randolph Page, M. D., New York........321

XVI.—Atrophic Rhinitis with Ozena—Its Etiology and Surgical Treatment. By Francis Patten Emerson, M. D., Boston........333

XVII.—Intranasal Treatment of Meckel's Ganglion. By E. M. Holmes, M. D., Boston........350

XVIII.—Bronchoscopic Removal of a Collar Button After Twenty-six Years' Sojourn in the Lung. By Chevalier Jackson, M. D., Pittsburg........360

XIX.—The Combined Laboratory and X-ray Indications for the Mastoid Operation. By George Sloan Dixon, M. D., New York........369

XX.—Ocular Manifestations in Nasal and Aural Diseases Which Probably Indicate Involvement of the Sympathetic Nervous System. By W. H. Haskin, M. D., New York....384

XXI.—Results in a Series of Cases of Tonsillectomy at the Massachusetts General Hospital, Three to Four Years After Operation. By J. Payson Clark, M. D., Boston........421

XXII.—Report of Removal of Fibromyoma Which Excluded the Auditory Canal; Hemophilia. By M. D. Lederman, M. D., New York........431

XXIII.—Abscess of Frontal Lobe of the Brain, of Otitic Origin, with Exhibition of Specimens. By T. Passmore Berens, M. D., New York........433

XXIV.—Report of the Committee Appointed to Consider the Best Methods to be Followed in the Teaching of Otolaryngology in Undergraduate and Postgraduate Schools. By D. J. GIBB WISHART, M. D., Toronto; S. MACCUEN SMITH, M. D., Philadelphia; CHAS. W. RICHARDSON, M. D., Philadelphia..453

XXV.—Suspension Laryngoscopy with Demonstration of Method. By WOLFF FREUDENTHAL, M. D., New York........464

XXVI.—The Training of the Specialist. By THOS. J. HARRIS, M. D., New York........475

XXVII.—Clinical Indications for the Mastoid Operation. By W. SOHIER BRYANT, A. M., M. D., New York........482

XXVIII.—Congenital Osseous Obstruction of the Postnasal Orifices. By C. W. RICHARDSON, M. D., Washington City..488

XXIX.—Intraocular Tension in Glaucoma Lowered by Injection of the Sphenopalatine Ganglion. By H. EDWARD MILLER, M. D., St. Louis........492

XXX.—Peritonsillar Phlegmon—Report of a Case. By F. E. HOPKINS, M. D., Springfield, Mass........496

XXXI.—Primary Tuberculosis of the Middle Ear. By CHAS. H. LONG, M. D., Chicago........499

XXXII.—Report of a Case of Temporosphenoidal Abscess of Otitic Origin—Operation—Recovery. By JAMES F. McCAW, M. D., Watertown........505

XXXIII.—Sinus Thrombosis Following Necrosis of Periosteal Flap. By OTTO GLOGAU, M. D., New York........509

XXXIV.—A Case of Paralysis of the Abducens Nerve Associated with Acute Mastoiditis. By HAROLD HAYS, M. D., New York........514

XXXV.—Diseases of the Labyrinth With a Report of Nineteen Cases. By FRANK R. SPENCER, A. B., M. D., Boulder, Colo........581

XXXVI.—Some Conditions Associated With the Loss of Cerebrospinal Fluid. By R. BISHOP CANFIELD, M. D., Ann Arbor........604

XXXVII.—Odd Cases of Nasal Deflection With Suggestion as to Treatment of Nasal Adhesions. By OTIS H. MACLAY, B. S., M. D., Chicago........623

XXXVIII.—The Operative Findings and Results in Mastoiditis, Acute and Chronic. By J. M. INGERSOLL, A. M., M. D., Cleveland 629

XXXIX.—Radiography as an Aid in the Diagnosis of Mastoid Disease. By FREDERICK M. LAW, M. D., New York.... 635

XL.—A Discussion of the Modus Operandi of Galvanic Nystagmus. By LESTER MEAD HUBBY, M. D., New York.... 638

XLI.—Exhibition of Radiograms Illustrating the Pathology of Mastoiditis. By HENRY S. BIRKETT, M. D., Montreal.... 649

XLII.—Further Observations on the Technic in the After-Care of the Radical Mastoid Operation. By PHILIP HAMMOND, M. D., Boston........ 652

XLIII.—The Faucial Tonsils as a Focus for Systemic Infection. By GEORGE E. SHAMBAUGH, M. D., Chicago........ 659

XLIV.—Report of Two Cases of Cerebrospinal Meningitis Presenting no Central Nervous Phenomena Until Shortly Before Death. By T. PASSMORE BERENS, M. D., New York.... 666

XLV.—Choice of Treatment in Chronic Suppurative Otitis Media. By GEORGE L. RICHARDS, M. D., Fall River........ 672

XLVI.—Radiographic Findings Illustrating the Anatomic Development of the Mastoid Bone. (Stereopticon Demonstration.) By WILLIAM H. STEWART, M. D., New York........ 677

XLVII.—Topography of the Tympanic Cavity. By JOHN A. CAVANAUGH, M. D., Chicago........ 699

XLVIII.—Some Anatomic and Clinical Relations of the Sphenoid Sinus to the Cavernous Sinus and the Third, Fourth, Fifth, Sixth and Vidian Nerves. By GREENFIELD SLUDER, M. D., St. Louis........ 725

XLIX.—Exhibition of Otitic Brain Abscess Cases and a Mastoid Exenteration by Nature. By S. MACCUEN SMITH, M. D., Philadelphia........ 769

L.—A Report of Three Unusual Cases of Stammering. By G. HUDSON MAKUEN, M. D., Philadelphia........ 776

LI.—Diagnosis and Treatment of Diphtheria. By JOSEPH B. GREENE, M. A., M. D., Asheville........ 782

LII.—Two Cases of Perforation of the Petrous Portion of the Temporal Bone Due to the Capsulated Bacteria. By H. B. GRAHAM, M. D., San Francisco........ 798

LIII.—The Surgical Management of Diseases of the Frontal Sinus. By ROBERT CUNNINGHAM MYLES, M. D., New York 807

LIV.—Report of a Case of Ulceration of the Larynx; Perichondritis of the Arytenoid Cartilages, Abscess and Partial Exfoliation of Both Cartilages Resulting from Typhoid Fever. By J. H. BRYAN, M. D., Washington, D. C........ 811

LV.—Vasomotor Disturbances of the Upper Air Tract. By C. W. RICHARDSON, M. D., Washington........ 817

LVI.—Histologic Pathology of the Accessory Sinuses. By JOSEPH C. BECK, M. D., Chicago........ 913

LVII.—The Endonasal Route of Attack in Hypophyseal Tumor Cases. By W. E. SAUER, M. D., St. Louis........ 971

LVIII.—Cavernous Sinus Thrombosis Due to Organisms of the Streptothrix Group. By HENRY HORN, M. D., San Francisco 989

LIX.—Suggestive Points of Analogy Between Otosclerosis and Arthritis Deformans. By JOHN F. O'MALLEY, F. R. C. S., London........ 1007

LX.—Complications That May Arise During or After the Submucous Operation for the Correction of Septal Deviations. By GEORGE W. MACKENZIE, M. D., Philadelphia........ 1020

LXI.—Aphthous Ulceration of the Upper Air Passages in Pulmonary Tuberculosis—A Retrospect and a Review. By JOHN N. MACKENZIE, M. D., Baltimore........ 1046

LXII.—Immediate and Remote Sequelæ of Radical Removal of the Faucial Tonsil—Observation of Two Hundred Cases. By VIRGINIUS DABNEY, M. D., Washington........ 1057

LXIII.—Decapitation of the Faucial Tonsil Followed by Digital Evisceration of Its Capsule. By J. A. ELLEGOOD, M. D., Wilmington 1067

LXIV.—Frontal Mucocele. By GEORGE W. MCCOY, A. M., M. D., Los Angeles........ 1073

LXV.—Report of a Case of Compound Follicular Odontoma of the Superior Maxilla. By L. W. DEAN, M. D., Iowa City 1077

LXVI.—Case of Meningitis in Which the Cisterna Magna Was Drained. By NORVAL H. PIERCE, M. D., Chicago...... 1081

LXVII.—The Use of Vaccines After the Mastoid Operation. By WILLIAM C. BRAISLIN, M. D., Brooklyn............1085

LXVIII.—On the Efforts of Nature to Cure Septic Thrombosis of the Lateral Sinus. By HARRY FRIEDENWALD, M. D., Baltimore 1090

LXIX.—Bone Conduction in Syphilis. By DR. OSCAR BECK, Vienna 1099

LXX.—The Nasal Voice With Reference to Its Bearing on the Practice of Rhinolaryngology. By ELMER L. KENYON, A. B., M. D., Chicago 1110

LXXI.—Caisson Workers' Deafness. By G. W. BOOT, M. D., Chicago 1121

LXXII.—The Ménière Symptom-Complex—A Clinical Review. By HILL HASTINGS, M. D., Los Angeles................1133

LXXIII.—Malignant Tumors of the Nasopharynx, With Report of Cases. By A. L. KELSEY, M. D., and J. M. BROWN, M. D., Los Angeles 1147

LXXIV.—Inflammation of the Lateral Columns of the Pharynx Leading to Abscess Formation, With Report of Cases. By HENRY L. SWAIN, M. D., New Haven................1156

SOCIETY PROCEEDINGS................228, 517, 820, 1166

New York Academy of Medicine, Section on Otology—228
239, 517, 525, 538, 820, 826

Chicago Laryngological and Otological Society—255, 558
834, 846, 1166, 1182

St. Louis Society of Medicine, Oto-Laryngological Section
271, 867, 870, 872

ABSTRACTS FROM CURRENT LITERATURE:

I.—EAR 873
II.—NOSE 889
III.—PHARYNX 893
IV.—LARYNX 895
V.—MISCELLANEOUS 902

BOOK REVIEWS................1197

ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY.

VOL. XXII. MARCH, 1913. No. 1.

I.

UPON THE PRESENT STATUS OF OTOSCLEROSIS.*

BY PROF. DR. ALFRED DENKER

HALLE, A./S.

The publications of the year just past, and especially the therapeutic suggestions which have been made, show us that we have by no means become united upon the conception of otosclerosis.

Before I enter into the discussion of this theme, it seems necessary for me to state clearly what I and my German colleagues understand by the term otosclerosis.

In my article on stapes ankylosis, which I read in 1903 before the meeting of the German Otological Society at Wiesbaden, and in my monograph on otosclerosis, I have set down for the conception of this disease the following definition:

We understand by otosclerosis a disease in which there is a permeable tube and a normal tympanic membrane, accompanied by a definite and characteristically marked clinical picture of a progressive difficulty in hearing, as shown by the functional tests. As a pathologicoanatomic foundation for the disease, investigations have revealed a loss of movement of the stapes, brought on by bony ankylosis in its framework or in the niche of the oval window, and a progressive spongification of the bony capsule of the labyrinth.

---

*Read before the Ninth International Otological Congress, Boston, August, 1912.

To this definition, upon the ground of our present knowledge, must be added that in addition to the disease of the bone, there is to be found, as shown by a histologic examination of a large number of cases, an atrophic degenerative process in the nerve endings in the membranous labyrinth.

Only those forms of disease which clinically and pathologicoanatomically correspond to the above definition will be dealt with, and not those forms of chronic affections accompanied by thickening of the interstitial connective tissue, which because of changes on the tympanic membrane are recognized as chronic adhesive processes or as the residues of long standing middle ear suppurations.

I understand thoroughly that the expression otosclerosis, which originated with v. Tröltsch, in no way corresponds to the pathologicoanatomic character of the disease. v. Tröltsch selected this name because he supposed that the otosclerosis led to an increased rigidity of the soft covering of the tympanic membrane, the hammer-anvil articulation, and especially of the window, which eventually led to a complete rigidity of the joint and to immobility of the stapes, in consequence of a calcification and ossification of the ligamentum annulare. He added, however, that anatomic examination would result in an increase in our knowledge of otosclerosis that would create for it a distinct position among ear affections. That this in reality is the case has been shown by the extensive literature in the last two decades describing the essential changes in the labyrinth capsule and the stapediovestibular articulation. We now know that lesions occur in the labyrinth capsule, in the ligamentum annulare, and in the footplate of the stapes which result in a clinical picture greatly resembling the symptoms appearing in middle ear disease. In a number of cases they absolutely correspond with the characteristic functional symptoms of middle ear disease, without demonstrable inflammatory changes in the soft coverings of the middle ear.

It has been repeatedly sought to substitute other expressions for otosclerosis. It has been proposed to designate the disease as "stapedioankylosis, with or without spongification of the labyrinth capsule." Heimann, in his article reported at Budapest, recommended the term "periostitis ossificans stapediovestibularis," and Manasse in his recent monograph calls this disease process "otitis chronica metaplastica of the labyrinth capsule." Apart from the fact that the expression "periostitis ossificans stapediovestibularis" does not include those cases in which the disease process surely arises primarily from the bony labyrinth capsule, I hold it as more correct to cling to the term otosclerosis until we are entirely clear as to its etiology and pathologic anatomy; and this, even to this day, in spite of the countless and careful works upon the subject, is still unknown.

Upon the grounds already mentioned, and in order to prevent the proposal of other therapeutic remedies which are beneficial in the treatment of chronic adhesive processes or the remains of a chronic middle ear suppuration, but which can have no feasible influence on a process affecting the labyrinth capsule or the stapediovestibular joint, I desire to propose that this German conception of otosclerosis be universally accepted.

In regard to the etiology of the pathologic process in the labyrinth capsule and the stapediovestibular articulation, there are two opposite opinions: some authors hold the opinion that the changes are to be considered as secondary ones, that they are caused by an inflammation of the membrane lining of the middle ear. Habermann particularly holds this view, upon the ground of thirteen cases which he examined, the results of which caused him to believe that the bone disease comes from an inflammation of the middle ear, whence it is transmitted along the larger vessels to the bone, and is disseminated by means of the medullary spaces. Katz considers, as does also Toynbee for some of his cases, the disease to be a definite inflammation of the stapediovestibular articulation, upon a rheumatic basis, and designates it as "arthritis rheumatica." A similar opinion is held by Schilling, who says that in his case he observed a spongification of the labyrinth capsule which took its origin from the periosteum of the middle ear. Scheibe also contends that in those cases in which an earlier middle ear suppuration has been demonstrated, but only for those, a causative relation between the two diseases is possible.

In contrast to this conception of an inflammatory middle ear disease as the antecedent cause, most of the remaining authors who have made histologic examinations contend that otosclerosis is a disease appearing primarily in the bone. Moss in 1867 was the first to express the opinion that in view of the lack of any change in the middle ear mucous membrane, the disease must be an ostitic process of the temporal bone.

Bezold and Scheibe, upon the ground of their histologic examinations, arrived at the belief that the bone disease took its origin either from the labyrinth capsule itself or from the periosteum of the niche of the oval window. The opinion that it is a primary bone disease of the labyrinth capsule is very energetically advanced by Politzer, who bases his views chiefly on the facts that in his large material most cases failed completely to show changes in the mucous membrane of the middle ear, and that the greatest changes were found in the deepest layers of the bone. This opinion is also held by J. Möller.

The pathologic anatomist Hanau, who examined the specimens from the cases published by Hartmann from the Siebenmann clinic, contends that the process at the stapes and the niche of the oval window is a hyperostosis with a rebuilding of the bone, and designates this newformed bone as metaplastic connective tissue bone, originating from the periosteum. Siebenmann, as the result of his investigations, concludes that the spongification does not arise from the periosteum, nor does it develop primarily from the labyrinth capsule, but that the oldest parts are to be found at the junction of the labyrinth capsule, which is formed out of the endochondrial substance, with the connective tissue bone arising secondarily from the periosteum (probably in the last named structure itself). Brühl also is of the opinion that we have to deal with a primary bone disease, which develops and progresses especially at the border between the endochondrial and connective tissue bone.

Hegener expresses as his opinion that in otosclerosis two distinct processes exist side by side. The bony change and the disease of the auditory nerve must be differentiated from each other, and the acoustic affection should not be considered as the result of the bony changes.

Manasse, in his monograph recently published, agreed with the opinion of Politzer and Siebenmann, that the pathologic changes which determine the clinical picture of otosclerosis are the product of primary disease of the labyrinth capsule. As to my own standpoint concerning the question of the point of origin of the pathologic changes in otosclerosis, Heimann and Fröschels, in their review, incorrectly stated that I consider otosclerosis a disease following a middle ear inflammation.

This is absolutely not the case. I have indeed granted the possibility that in those cases of otosclerosis accompanied by inflammatory changes in the mucous membrane of the middle ear, the disease might have taken its origin from the mucosa. But I further distinctly stated that this conception of the etiology of the disease leaves entirely unexplained the origin of the bone process in the labyrinth capsule in all cases in which a normal middle ear mucous membrane is demonstrated.

For these cases, only a primary disease of the bone or the periosteum can enter into consideration. That the bone changes can occur without cooperation of the periosteum, we are justified in believing, following the publications of Siebenmann, Politzer and Manasse, who found isolated foci in the cochlea which nowhere involved the middle ear mucous membrane. It is therefore certain that in a number of cases the pathologic changes take their origin absolutely from the bony labyrinth, while the middle ear mucous membrane cannot with the same certainty be designated as the originating point. It cannot, however, be excluded that in some cases the disease process extends from the mucosa of the middle ear into the bone; but it is just as possible that the mucous membrane changes found in otosclerosis are accidental, and it is not necessary to bring it into consideration as the originating point of the disease.

There prevails also no unity in the interpretation of the bone alteration. One group of authors holds the inflammatory character of the change as proven. Manasse especially holds this opinion, and upon the ground of his histologic investigations makes the following statement: "The commencement with the formation of osteoid and granulation tissue, the interchange between apposition, resorption and renewed apposition, the giant cell formation, these are all processes such as we find in chronic inflammatory processes in the bone—for example, in ostitis chronica fibrosa. There also occur the conversion of richly cellular marrow into fibrous marrow, the production of a hard, compact bone, and exostosis formation, all characteristics of a productive process analogous to chronic inflammation in other organs." Manasse proposed, therefore, for the entire disease process in the bone the name "ostitis chronica," and since the characteristic feature of this inflammatory process is the change in the old labyrinth capsule, he called the entire affection "ostitis chronica metaplastica of the labyrinth capsule."

Brühl designates the bone changes in the labyrinth capsule, in agreement with Orth, as a spongy hyperostosis, and explains their origin as follows: By means of the vessels of the hyperplastic periosteum, and at the same time of the vessels of the Haversian canals of the periosteal built bone, a dissolving of the old bone is brought about, which is followed by a regular, even, excessive formation of connective tissue bone.

Siebenmann comes to another conclusion, after careful study of a large number of temporal bones. According to him, the beginning of the progressive spongification of the labyrinth capsule occurs not, as Manasse supposes, in an extension through new bone, but in the inward growth of cells from the periosteal tissue through the bone covering into the normal Haversian canals of the labyrinth capsule. Lacunar resorption from the walls, with the aid of mono- and polynuclear osteoblasts, goes hand in hand with it and fails in no case. New bone apposition results only where resorption processes have taken place. The newly apposed bone contains more chalk than the old bone, and does not deserve to be known as osteoid substance. As a differentiation from ostitis fibrosa, the spongifying process of the labyrinth capsule occurs without inflammatory symptoms. Granulation tissue, leucocytes and plasma cells are not present.

Since our colleague, Dr. Siebenmann, intends to demonstrate his newest specimens after my address, you will have the opportunity to judge for yourselves the correctness of the Siebenmann theory. But when we have arrived at a decision concerning its inflammatory or noninflammatory character, there remains still the questions: What is the essential cause? What gives the impetus to these alterations in the labyrinth capsule? Concerning this, also, there have been many hypotheses suggested. For a long time the authors have observed that otosclerosis is a disease which occurs more frequently in women than in men. Although Walb, as a reason for this phenomenon, suggests that the affection prefers the femal sex because in them is observed more frequently bad conditions of the blood and distinct anemia than in the male sex, which conditions he claims are the etiologic factors in otosclerosis, it is more probable that pregnancy and the puerperium are answerable for this striking property of the sex. This opinion has much more value, when we remember that a large portion of these otosclerotic women trace the beginning of their difficulty in hearing to the time of pregnancy or the following period of convalescence.

The coincidence of pregnancy with the beginning of the otosclerosis suggests a causal relationship between the function of the hypophysis cerebri and the ear disease. It is well known that the hypophysis regularly enlarges during pregnancy and returns again to normal during lactation. We suppose that the hypophyseal changes during pregnancy depend upon a diminished function of the ovary. This conclusion is reached because the same hyperplasia of the hypophysis occurs in experimentally castrated female and male animals.

Furthermore, it has been possible to prevent or to limit the hypophyseal hyperplasia by feeding with extract of the ductless glands. We know, furthermore, that as a result of increase or disturbance of the internal secretion of the hypophysis, we find bony changes which are called acromegaly. In this disease are found exostoses and irregular porosity of the external layer, whose histologic examination shows that this is not a specific pathologic process, but that apposition and resorption take place exactly in the same manner as in normal bony growth. The causal relationship between the anomaly of the hypophysis and acromegaly is shown by the fact that the latter can be cured by operation for hypophyseal tumors.

Although the changes in the labyrinth capsule in otosclerosis are not analogous to the bony changes of acromegaly and the puerperal bony processes, still the fact that the bony changes for which the hypophysis is responsible and the beginning of otosclerosis both occur during pregnancy, gives food for thought, and I would like to call your attention to the possibility of an etiologic connection between malfunction of the hypophysis and the origin of otosclerosis, especially in persons with an hereditary taint. It would be a very good plan to examine cases of hypophyseal tumors, with or without acromegaly, for symptoms of otosclerosis. However, even if we are willing to concede that the cases of otosclerosis which begin at the time of pregnancy can be caused by lesion of glands with internal secretion, only a portion of the cases can be thus explained. Probably constitutional anomalies may cause the disease. This view is supported by the fact that the disease is bilateral and the degree of deafness in many cases is the same on both sides. This similarity, in many cases, is represented on the pathologicoanatomic side by a complete symmetry of the bilateral bony changes, which caused Otto Mayer and Manasse to believe that a disease of the vascular system is the cause of the affection.

Undoubtedly, the most important etiologic factor is heredity. Although Panse could find in forty patients with synostosis of the stapediovestibular articulation only three with deaf brothers or sisters, Bezold, Denker and Siebenmann demonstrated in their cases of sclerosis percentages of 52, 40 and 35. The family trees of otosclerotics published by Körner and Hammerschlag are of special interest in the question of inheritance of the disease. Körner thought that not the disease itself but the predisposition to the disease was inherited, and believed that in otosclerosis it is always a case of inherited predisposition thereto. The explanation of the fact that we cannot in all cases show heredity connotes that the determinants representing the special peculiarity do not necessarily become active in each offspring, but can skip one or more generations and suddenly reappear. For these reasons, Körner agrees with Siebenmann that in otosclerosis there is an abnormal postembryonic developmental process.

Hammerschlag agrees with Körner that there is always an inherited predisposition in otosclerosis, but concludes that the primary appearance of otosclerosis is not to be sought among the lower mammals, but in the primates. He is convinced that if the disease ever appeared among the primates, a primary appearance among them is possible today. It is unnecessary to explain the apparently spontaneous appearance of cases of otosclerosis as cases of latent heredity. In his work, Hammerschlag gives two family trees in which both deafmutes and otosclerotics appear, and calls attention to the possibility of a genetic connection between these two aural lesions. As a basis for this, he states that in many cases of congenital deafness, the same bony lesions are found as in otosclerosis, and that in the latter degenerative atrophic changes in the membranous labryinth are frequently found which correspond to those found in congenital deafness. He agrees with Hegener that in otosclerosis there is not a secondary process in the acusticus, but a primary one, coincident with and independent of the bone lesions. Often the disease in the nerve appears before that in the bone. Among other proofs is that of the subjective noises frequently appearing earlier.

On the grounds of his investigations upon deaf cretin children and on a deaf cretin dog, Alexander comes to the conclusion that otosclerosis is a congenital disease. Since the bony changes found in his cases completely agree histologically with those in the labyrinth capsule in otosclerosis, he is convinced that the latter disease is also a congenital one. He calls the lesion "ostitis vasculosa," and believes that the otosclerotic has very vascular bony foci in his temporal bones at his birth and even during his embryologic period, and that only at puberty, and in some cases much later, do they extend to the endosteum of the labyrinth or lead to fixation of the stapes, changes on the cochlear window and secondary degenerative processes in the nerve endings.

Brühl agrees with Alexander that the hereditary predisposition to the disease probably has its anatomic basis in a congenitally misplaced spongification focus in the labyrinth capsule, which begins to develop under the influence of certain stimuli. The changes which affect the vestibular articulations and their vicinity are probably due to a formative stimulation of bone and new formation consisting of pressure and traction which have their greatest action, on anatomic grounds, just at the anterior circumference of the vestibular window, the site of predilection for changes in stapes ankylosis. The interrupted traction on the bone caused by movement of the footplate of the stapes, sets up an irritation in the bone and periosteum. In addition, the continual contraction of the musculus tensor tympani, which likewise lies just at the anterior circumference of the stapes, causes a permanent unrest in the periosteum and bone of this region.

Brühl's hypothesis is opposed by O. Mayer. He believes that it is incomprehensible how the atypical cases (unconnected with the tympanic mucous membrane) in which stapes ankylosis is present should be due to traction on the annular ligament, and the formative stimulus should cause disease of the bone at remote places, while in the cases where there is no stapes ankylosis, the same foci should be due to some other cause. The fact that in many cases the lesions in the labyrinth capsule show a complete symmetry, led Mayer to the belief that these foci correspond to blood vessels and are caused by their disease. The causes of such lesions seem to be above all disturbances in the vasomotor functions, but he thinks that other causes of local circulatory disturbances, especially organic disease of the blood vessels (arteriosclerosis), may cause the same changes. According to him, the heredity consists in an inheritance of the cause of the disease, for it is well known that heredity plays a great role in both arteriosclerosis and vasomotor neuroses. That Siebenmann considers otosclerosis to be a congenital condition, has already been noted above.

While the other authors could accept the heredity of otosclerosis only on the grounds of the history, and the subjective and objective findings, Manasse was able to show anatomically the typical bony changes in two members of the same family. There seems to be no doubt, therefore, that otosclerosis is actually an inheritable disease.

Of the other etiologic factors, lues, above all, has been accused of being the cause of the disease. Habermann made the most thorough study of this phase, and is convinced of the causal relationship between otosclerosis and syphilis. Later investigations have shown, however, that lues is not a frequent etiologic factor in producing the characteristic changes in the labyrinth capsule—first, because the vast dissemination of syphilis would cause a more frequent occurrence of the aural affection; and second, because men suffer from syphilis much more frequently than women, while otosclerosis is specially a disease of the female sex. But especially do the results of the Wassermann reaction argue against the correctness of Habermann's theory: the examination almost invariably gives results which speak against a luetic etiology.

Many authors (Panse, Hartmann, Siebenmann, etc.) have noticed that many patients refer their disease to a severe cold. Although we have no exact idea of the manner of action of low temperatures, we cannot reject the possibility of some such etiology, especially since very observant patients have noticed a permanent exacerbation of their trouble following considerable cooling off. Perhaps it is disturbance transmitted through the vascular system.

That a trauma may lead to stapes ankylosis with the functional symptoms of otosclerosis was shown by a case published by Politzer in 1862. If we review what has been written concerning the etiology of otosclerosis, it seems correct to state that in the majority of cases, at least, we have to do with a hereditary anlage as the cause of the disease. This predisposition lays the foundation upon which, under the influence of certain stimuli, there arises the affection with its characteristic symptoms and pathologicoanatomic changes. These stimuli are the increased bony formation during puberty and the bony changes during pregnancy and the puerperium, which are probably dependent on the hyperplasia of the hypophysis, which appears during pregnancy. Furthermore, I believe in the correctness of Otto Mayer's claim, that disturbances in the circulation such as are present in arteriosclerosis, vasomotor neurosis, and lues may give the impetus to the development of the disease. It has not yet been proved that the continuous movement of the stapedial footplate and the permanent contraction of the musculus tensor tympani can be regarded as a cause for the fact that the bony alterations develop by predilection at the anterior circumferance of the vestibular window, as Brühl claims.

Shortness of the time at my disposal forbids me to go into an extended discussion of the pathologicoanatomic changes in the labyrinth capsule, the ligamentum annulare, and the stapedial plate. I must content myself with demonstrating the characteristic pictures with the projection apparatus, at the close.

As to the symptoms and the clinical course of the disease, I will be brief, since the greater part is well known. You know that frequently subjective noises of the most varied kinds (roaring, ringing, knocking, etc.) and of varying intensity are the first indications of the disease, and that these noises are sometimes so severe and act so unfavorably upon the patient, that they are regarded by them as a greater evil than the deafness. According to the statistics of Bezold, Politzer and myself, they are observed in 70 to 80 per cent of cases of otosclerosis.

The diminution of hearing usually appears very gradually, so that the patient sometimes cannot tell when it began. Often only one ear is affected, and the other not until later. The course is usually insidious, and frequently many years pass before the deafness becomes very great. However, frequently rapid exacerbations are seen. As to the degree of deafness and the results of exact functional tests, they are dependent on the location and extent of the changes in the capsule, the annular ligament, the footplate and the membranous labyrinth. The variability of these lesions and the resulting symptom complex cause the disease to be divided into three forms:

(1) The pathologic changes are located exclusively at the vestibular window and its vicinity, and have led to a bony fixation at the footplate—isolated stapes ankylosis.

(2) Appearance of multiple spongifying foci in the labyrinth capsule, with atrophic degeneration of the membranous labyrinth without stapes ankylosis.

(3) Bony fixation of the stapes plate, combined with other foci in the labyrinth capsule and with atrophy of the membranous labyrinth.

If it is a case of purely stapes ankylosis (group 1), whisper is usually heard at a short distance from the ear. Numbers 9 and 5 (German) are heard the poorest. By an exact examination we can obtain the symptom complex known as Bezold's triad, characterized by raising of the lower tone limit, lengthening of the bone conduction in the Weber-Schwabach test, and negative Rinné. The upper tone limit is usually normal or very slightly lowered.

If there are isolated foci in the labyrinth capsule, and if an atrophic degeneration of the membranous labyrinth results from the extension of these foci to the endosteum of the cochlea, or from other causes (group 2), without changes in the stapediovestibular articulation, the functional symptoms of nerve deafness appear. Whisper is not heard in many cases, and loud tones are often heard only at a short distance. The perception is worst for words containing high sounds (6, 7). Although the lower tone limit can be about normal, the upper is greatly lowered. Bone conduction (Weber-Schwabach) is shortened and Rinné is positive.

In the combination of stapes ankylosis with spongifying foci in the labyrinth capsule and atrophy of the membranous labyrinth (group 3), the following functional symptoms are observed: Hearing distance for speech and whisper greatly reduced; the lower tone limit is raised with a simultaneous lowering of the upper; bone conduction is not increased and in many cases is decreased, while Rinné is not negative, but briefly positive and even normal. We have, therefore, functional symptoms which speak neither for an uncomplicated affection of the conduction apparatus nor for a pure disease of the internal ear. In such cases we must use the other methods of examination, which will be mentioned in discussing the diagnosis.

Disturbances of equilibrium and attacks of dizziness of different degrees are frequently observed in otosclerosis. They may be referred to a direct or indirect change in the vestibular nerve or its endings in the vestibule and ampullæ. Siebenmann proposed the theory that pressure and density changes which the labyrinthine fluid undergoes through the influence of the spongification extending to the endosteum of the labyrinth, are responsible for the lesion of the function of the nervus cochlearis as well as disturbances of equilibrium. He thinks that the newformed spongiosa can break through into the perilymph, and by means of the sudden increase of intralabyrinthine pressure and changes of position cause Meniere's disease, both in its milder forms and in the form of apoplectic attacks of vomiting, nystagmus and falling. In the same way Siebenmann explains the sudden appearance of loud subjective noises, such as thunder, popping, shooting, etc.

Pain in the depths of the ear is relatively seldom observed in otosclerosis, and is to be referred to changes in the periosteum or the stapediovestibular articulation, or to changes in the sensory fibers of the nervus facialis. The patients comparatively frequently complain of a feeling of pressure or tension in the ear.

The hyperemia of the promontorial mucous membrane, which Schwartze claimed is the characteristic symptom of otosclerosis, has been observed both in nerve deafness (Siebenmann) and in normal hearing (Brühl), although rarely. These cases, however, may be initial symptoms of cases of otosclerosis, which later develop into a severer form, and we can regard promontorial hyperemia as a symptom which, if not absolutely certain, is at least one which, with others, aids in the diagnosis of otosclerosis.

The paracusis Willisii, so often observed in this disease, was explained by Willis himself as due to a slack drum. Toynbee explained it as a result of a shaking of the chain of ossicles, and Politzer also thought that the ossicles whose articulations had become rigid had by this shaking been displaced from their fixed positions and thus made suitable for conducting sound. Siebenmann gives the following explanation. He claims that the otosclerotic is disturbed less by street noises, such as railway journeys, than the normal hearing individual, because they are composed of lower tones, the perception of which he has lost. To this is added the fact that normal hearing people speak louder in loud noises, and for this reason the otosclerotic can apparently hear loud sounds better than in quiet places. Urbantschitsch explains the paracusis Willisii by an increased function of the nervus acusticus.

A symptom observed frequently by patients with nerve deafness as well as by otosclerotics, to which Urbantschitsch and Walb have drawn attention, is the decrease in hearing, the rapid weariness following strained attention.

Recently Fröschels described a symptom consisting of a diminution of the tickling sensation parallel to the loss of hearing. If a small probe armed with a cotton brush is introduced about 1 cm. into the meatus and moved gently to and fro, it will often be found that the tickling sensation is less developed in the worse ear. In this test it is of course understood that no suppuration exists or has existed, since this also disturbs the sensibility.

The diagnosis of otosclerosis is easy in those cases where the process is confined to the stapediovestibular articulation and its vicinity (group 1). If the drum is normal, and if catheterization does not improve the hearing, while showing the permeability of the tube, we can say that it is a case of stapes ankylosis due to otosclerosis, when we find a distinct loss of hearing and the symptom complex known as Bezold's triad (raising of the lower tone limit, lengthening of bone conduction, negative Rinné). The cases where the diagnosis was made on these findings invariably showed its correctness whenever they came to postmortem.

The diagnosis is more difficult when the disease is not confined to the vicinity of the vestibular window, but affects the whole labyrinth capsule to a greater or less extent (group 2). If there is not complete or a high degree of deafness due to degenerative atrophy in the labyrinth, we can usually make the differential diagnosis between nerve deafness and a combination of it with stapes ankylosis. For this we use the test described by Gelle. If the Rinné is positive, and there is a marked raising of the lower tone limit, and if Gelle's test is negative—if compression of air in the meatus has no effect upon the intensity of the tuning fork's note—it is very probable it is a case of otosclerosis, i. e., stapes ankylosis combined with extensive spongification of the labyrinth capsule and atrophy of the membranous labyrinth.

In addition to the above, we can use in such cases a test described by me. If doubtful cases are tested with a continuous series of tuning forks as to the duration of their perception, it will be possible to demonstrate in otosclerosis that the part of the scale still heard will have its notes heard by air conduction longer in proportion to the number of vibrations, which is not the case in pure nerve deafness. If by this test we obtain a curve which in general is an ascending one and shows only unimportant deviations, we can with almost complete confidence make the diagnosis of stapes ankylosis complicated by an involvement of the internal ear. If, however, the test gives a very irregular line, it is an exclusively labyrinthine affection, i. e., a purely nerve deafness.

On account of what has been written, I cannot share the skepticism of Panse and Manasse, who believe it is impossible in very many cases to make the diagnosis of otosclerosis. It must of course be admitted that the small isolated bone foci in the labyrinth capsule cannot be found intra vitam, and that extensive disease of the labyrinth capsule without stapes ankylosis cannot be diagnosed from nerve deafness by functional tests. The other cases, however, can be confirmed by the functional tests, bearing in mind the anamnesis and objective findings. It must be remembered that most cases of otosclerosis are found between the ages of twenty and forty, while nerve deafness usually does not appear until after forty.

In the treatment of otosclerosis, this prophylactic consideration must be remembered. Since experience has taught us that exacerbations of otosclerosis follow pregnancy and the puerperium, the prevention of conception is strongly urged. Furthermore, consangineous marriages are to be forbidden, since the offspring are often deaf or very hard of hearing. They are especially to be forbidden if the parents already show a distinct taint of otosclerosis. The otosclerotic, furthermore, must beware of "catching cold," although we cannot explain just how a rapid cooling off of the body has any influence on changes in the labyrinth capsule. Probably it is an influence acting through the vascular system. Finally, the otosclerotic must beware of cold douches upon the head, and sea voyages, since experience has shown that the disease is made worse thereby. As to the active treatment of manifest otosclerosis, I regret to say that I have the same viewpoint today that I expressed in my monograph. It is, that because of the kind of osteopathologic process, a therapy based on mechanical action or local operative treatment will have only a slight effect or none at all. This kind of treatment is justified in chronic adhesive processes and in the results of chronic suppuration of the middle ear, but not in otosclerosis.

All treatments directed toward mobilizing or extracting the stapes have resulted in so small a permanent improvement in the hearing that they are not to be recommended, especially since there is a possibility that they may make the disease worse.

When there are subjective noises, we have seen more or less permanent result in some cases from the use of the electric drum masseur, after catheterization and pressure probing proved useless. Since these subjective noises are often especially troublesome in otosclerosis, I think we are justified in using this massage treatment repeatedly, even when the result obtained is only transitory.

For internal treatments, Politzer prescribes potassium iodid in doses of 1 gram daily, and he believes that it is possible in some cases to check thereby the further progress of the case. He recommends treatments of ten to fifteen days, in intervals of two months, four or five times a year.

The favorable results which Vulpius, Brühl, Alt and Eitelberg obtained by thyroid treatment have not been confirmed by others.

I have had no experience with the transtympanal electroionization recommended by Malherbe, but I cannot conceive how such a treatment would have any effect on a bone process in the labyrinth capsule. It seems to me to be doubtful also whether the results which Malutin claimed he obtained with mud baths will be confirmed by others. Furthermore, the fibrolysin treatment, with which Erbstein obtained a slight improvement in the hearing of one out of four cases, does not seem to me to offer much hope. Whether the diplococcus serum recommended by Ferreri can have any effect in the disease of the labyrinth seems to me very questionable. The experiments of Müller with the serum antisclereux Malherbe had no special result.

More is to be expected, according to my experience, from the internal administration of phosphorus, as recommended by Siebenmann. Upon the basis of experiments by Mirwa and Stotzner, which showed that phosphorus, when rationally used, is able to prevent the formation of the normal spongiosa, at least in long bones, and to favor the production of compact bone, Siebenmann gives daily one to two tablespoonfuls of Kassowitz's phosphorus emulsion, to check the spongifying process in the labyrinth capsule. For many years I have in my clinic treated otosclerosis with phosphorus, for the last three years in the form of phytin (phytin 0.25 in original tablets, two or three daily, after meals), which is well borne in most cases. Although we cannot expect too much from this therapy, still I think we can give patients the hope that the process will be checked and that the subjective noises will be favorably influenced, perhaps made to disappear. Of course, we cannot be sure whether the improvement is not to be often referred to a favorable influence of the drug upon the general condition of the body and of the nervous system.

As I stated above, it seems to me that there is a possibility of a causal relation between otosclerosis and improper function of the hypophysis. For this reason, in my clinic we have begun to prescribe hypophysin or pituitrin, especially since Moraczewski's experiments have shown that feeding with hypophysin tablets prevents the elimination of phosphorus. According to v. Fränkel-Hochwart and Frölich, the hypophyseal preparation is practically innocuous. The hypophysin is prescribed in tablets (two or three daily).

Since the enlargement of the hypophysis which regularly occurs during pregnancy has a possibly causal connection with the origin or exacerbation of otosclerosis, which is often noted during this period, we may try in the case of women with hereditary taint to prevent the hypophyseal hypertrophy or to restrict it by exhibition of extract of sexual glands. That this can be done, has been noted above. We prescribe ovarian tablets (Merck) or spermin tablets (Poehl), two or three daily.

As to otosclerol, which has recently been recommended, and which has yielded good results as far as tinnitus is concerned, I have not had sufficient experience. Likewise, I have not had sufficient opportunity to test the value of vasotonin, which has very recently been recommended for otosclerosis.

If the disease is bilateral, and the hearing is greatly reduced, so that intercourse with people is greatly hindered, it is necessary strongly to urge the patient to take a course in lip reading, in order that the eyes may at least partially assist the hearing.

(Demonstrations of photograms of specimens of otosclerosis (Katz, Bezold-Scheibe, Politzer, Siebenmann, Habermann, Brühl) with the projectoscope.)

II.

THE REPORT OF AN EXAMINATION OF BOTH TEMPORAL BONES FROM A HUNDRED AND TWENTY INDIVIDUALS IN REFERENCE TO THE QUESTION OF SYMMETRY IN HEALTH AND DISEASE.

By Arthur Cheatle, M. D.,

London.

PREFATORY NOTE

The examination was undertaken to ascertain facts as to the symmetry of the temporal bones, a subject on which little reference is made in otological and anatomical literature, but which has, I venture to think important bearings on the causation, diagnosis and treatment of disease.

The following is a list of the sex and age of the individuals:

1. M. $\frac{9}{12}$.
2. F. $1\frac{7}{12}$.
3. ?. $1\frac{9}{12}$.
4. M. 2.
5. F. 2.
6. ?. $2\frac{2}{12}$.
7. M. $3\frac{3}{12}$.
8. F. 6.
9. M. 9.
10. M. 11.
11. F. 16.
12. M. 20.
13. F. 21.
14. F. 22.
15. M. 24.
16. M. 24.
17. F. 25.
18. M. 28.
19. M. 28.
20. F. 28.
21. F. 28.
22. F. 29.
23. M. 30.
24. M. 30.
25. M. 30.
26. F. 30.
27. M. 33.
28. M. 33.
29. M. 33.
30. F. 33.
31. M. 34.
32. M. 35.
33. F. 35.
34. M. 36.
35. F. 36.
36. F. 36.
37. M. 37.
38. M. 37.
39. F. 37.
40. M. 38.
41. M. 38.
42. F. 38.
43. F. 38.
44. M. 39.
45. M. 39.
46. M. 39.
47. M. 39.
48. M. 39.
49. F. 41.
50. M. 42.
51. F. 42.
52. M. 43.
53. F. 43.
54. M. 44.
55. M. 45.
56. M. 45.
57. M. 45.
58. M. 47.
59. M. 48.
60. M. 48.
61. F. 48.
62. F. 48.
63. M. 49.

64.	M.	49.
65.	M.	49.
66.	F.	49.
67.	M.	50.
68.	F.	50.
69.	F.	50.
70.	M.	51.
71.	M.	51.
72.	M.	51.
73.	M.	52.
74.	M.	53.
75.	F.	53.
76.	F.	54.
77.	F.	54.
78.	F.	55.
79.	F.	55.
80.	F.	55.
81.	M.	56.
82.	F.	56.
83.	M.	57.
84.	F.	57.
85.	M.	58.
86.	M.	58.
87.	M.	59.
88.	M.	59.
89.	M.	59.
90.	M.	59.
91.	M.	60.
92.	M.	60.
93.	M.	61.
94.	M.	61.
95.	F.	61.
96.	F.	61.
97.	F.	61.
98.	F.	62.
99.	F.	62.
100.	F.	63.
101.	M.	64.
102.	M.	64.
103.	F.	64.
104.	F.	64.
105.	M.	66.
106.	F.	66.
107.	M.	67.
108.	M.	68.
109.	M.	68.
110.	M.	71.
111.	M.	72.
112.	M.	72.
113.	F.	72.
114.	F.	73.
115.	F.	76.
116.	F.	77.
117.	F.	81.
118.	M.	82.
119.	F.	86.
120.	F.	89.

I regret the deficiency in the number of infants and children, but this part of my collection is still in its infancy. The sex is known in all except in two, one aged one year and nine months and the other two years and two months. There are sixty-eight males and fifty females. The specimens are arranged according to age, and if there are several of the same age, the males are placed before the females. Each bone is separately placed and numbered, the right immediately preceding its corresponding left bone; the odd numbers therefore represent the right and the even numbers the left bones.

In referring to specimens the following method is used:

$\frac{\text{Number of the right bone}}{\text{Number of the left bone}}$, sex, age; for example, $\frac{71}{72}$ F. 36.

The bones are, in the main, vertically sectioned through the outer antral wall, mastoid process, middle-ear tract and labyrinth. Sections in other directions have been made when thought necessary. Further sections will be made immediately if any members of the Congress express such a wish.

The report is considered under two main headings: (A) In health. (B) In disease.

A. In Health

This part is considered under two headings: (1) The Exterior; (2) the Interior.

1. THE EXTERIOR

(i) *The external surface*

Symmetry is the rule in markings, shape and size.

(*a*) In one specimen *the temporal ridge* behind the antrum is more prominent on the right side, $\frac{161}{162}$ M. 56.

(*b*) In two sets the *posterior zygomatic line* on the right side above the antrum has an inclination downwards and backwards and an overhanging appearance associated with a markedly high-lying antrum, conditions which are not seen on the left side and in which the antrum is not high lying, $\frac{147}{148}$ M. 53 and in $\frac{185}{186}$ M. 61.

(*c*) *A specially large foramen*, evidently from a vessel, is present immediately above the meatal spine, leading to the coarse cells in the outer antral wall on both sides in $\frac{165}{166}$ M. 57.

(*d*) *Perforations in the cortex of the supra-meatal triangle* exposing the cells lining the outer antral wall are present, on the right side only in $\frac{73}{74}$ M. 38 and on the left side only in $\frac{39}{40}$ F. 25. There is no evidence of disease in either.

(*e*) *The supra-meatal spine* presents many variations and one side is occasionally larger than the other, but symmetry is the rule.

(*f*) The remains of the *masto-squamosal suture* when present are usually symmetrical, but in three sets one side is more marked than in the other; one on the right side, $\frac{207}{208}$ F. 64, and two on the left, $\frac{29}{30}$ M. 24, $\frac{213}{214}$ F. 66.

(*g*) There is very little *variation in the size and shape of the mastoid process*; the left is slightly larger and more pronounced in $\frac{7}{8}$ M. 2 and the right in $\frac{185}{186}$ M. 61 and $\frac{87}{88}$ F. 41, where the right cellular mastoid is rounder than the diploëtic left. Even in sets which vary strikingly in their interior there is no striking external variation, as in $\frac{163}{164}$ F. 56, $\frac{193}{194}$ F. 61, $\frac{227}{228}$ F. 73, in all of which one bone is cellular and the other is diploëtic.

In connection with this, Iwanoff (Politzer's text-book, page 39) states that in brachycephalics the right mastoid process is poorly developed.

(*h*) *Thinning and perforation of the anterior surface of a cellular mastoid process*, close to the tympanic plate, is seen on the left side in $\frac{117}{118}$ M. 48. The condition is not due to disease in the mastoid cells, and would have passed without comment if precisely the same thing had not been seen in the left bone of a man, age forty-five, not appearing in this series.

(*i*) *A large and unusually situated external opening for the petro-squamosal sinus* between the base of the zygoma and the post-gleniod tubercle is seen on the right side only in $\frac{17}{18}$ M. 9.

(*j*) The variations in the opening of the mastoid emissary vein will be considered when dealing with the posterior surface.

(*ii*) *The superior surface*

(*a*) *Grooving for the petro-squamosal sinus* is very well marked in three sets, although remains may be seen in others. There may be marked asymmetry.

$\frac{1}{2}$ M. $\frac{9}{12}$, the grooving is seen at the back part, that on the left side being more marked.

$\frac{13}{14}$ M. $3\frac{3}{12}$, the grooving is well marked on both sides, but is larger and deeper on the left. There is an external opening on both sides, the left being the larger, in the most usual position between the post-glenoid tubercle and the tympanic plate. On the right side the sinus passes under a bridge of bone before opeing into the lateral sinus, while on the left side it is an open groove all the way.

$\frac{17}{18}$ M. 9, the grooving is large and deep all along the line of suture on the right side, the sinus passing under a bridge of bone behind to open into the lateral sinus groove; in front it perforates by a large opening between the base of the zygoma and the post-glenoid tubercle—a rare position. On the left side there is no grooving, the only existing sign being an external opening at the outer extremity of the Glaserian fissure.

$\frac{121}{122}$ F. 48, the grooving is present on the left side only. there is no external opening.

(*b*) *Dipping down of the middle fossa,* altering the shape of the antrum by depressing its roof or causing the dura mater to intervene between the upper part of the cavity and the outer surface, occurs in nineteen sets. It is never symmetrical. It is present on both sides in six, but always more on one side than the other.

On both sides, but more marked on the right:
1. $\frac{167}{168}$ F. 57.
2. $\frac{193}{194}$ F. 61.
3. $\frac{209}{210}$ M. 66.
4. $\frac{227}{228}$ F. 73.

On both sides, but more on the left:
5 $\frac{199}{200}$ F. 63.
6. $\frac{203}{204}$ M. 64.

On the right side only in six:
1. $\frac{33}{34}$ F. 25.
2. $\frac{37}{38}$ M. 28.
3. $\frac{45}{46}$ M. 30.
4. $\frac{51}{52}$ F. 30.
5. $\frac{111}{112}$ M. 45.
6. $\frac{163}{164}$ F. 56.

On the left side only in seven:
1. $\frac{73}{74}$ M. 37.
2. $\frac{93}{94}$ M. 39.
3. $\frac{121}{122}$ F. 48. Depression behind the antrum.
4. $\frac{161}{162}$ M. 56.
5. $\frac{187}{188}$ M. 61.
6. $\frac{189}{190}$ F. 61.
7. $\frac{231}{232}$ F. 77.

It will be seen that it occurs about equally in the two sexes, nine being males and ten females.

It cannot be stated that the type of bone as regards presence or absence of cells has any influence in its production, for it is seen in all types. There is no external sign of its presence apart from cases of hydrocephalus which is not here represented.

The antrum may be high lying and yet the middle fossa may dip down, shutting part of the cavity out from the surface, as on the right side of $\frac{111}{112}$ M. 45.

(*c*) *A deep groove is present in the neighbourhood of the hiatus Fallopii on* the left side only in $\frac{95}{96}$ M. 39, and on the right side only in $\frac{233}{234}$ F. 81; it is apparently for a vessel.

(*iii*) *The anterior surface.*

(*a*) *The openings for the petro-squamosal sinus* have already been alluded to.

(*b*) *The opening in the tympanic plate* is closed by bone on both sides at an early age in $\frac{9}{10}$ F. 2. On the other hand they are both unclosed by bone in $\frac{25}{26}$ F. 21 and $\frac{83}{84}$ F. 38. In $\frac{13}{14}$ M. $3\frac{3}{12}$ the opening is closed on the left side and not on the right.

(*c*) Sometimes, by no means always, above the age of sixty years *the tympanic plate undergoes marked thinning*, and a gap closed by membrane only appears in the atrophied bone, closely resembling the condition seen in early life. It is apparently due to movements of the jaw. It may be seen on both sides in—

$\frac{225}{226}$ F. 72.
$\frac{237}{238}$ F. 86.

On the right side in—
$\frac{235}{236}$ M. 82.

On the left side in—
$\frac{205}{206}$ F. 64.

(*d*) *Bony spicules crossing the floor at the bend of the internal carotid canal* are seen on the left side of $\frac{31}{32}$ M. 24. They are not present on the right side, but there is a small groove separated from the canal floor by a slight ridge.

(*iv*) *The posterior surface*

(*a*) *The lateral sinus.* As is well known, the right lateral sinus is usually larger and more forward than the left whatever the type of bone, owing to the opening of the longitudinal sinus being usually towards that side, and as Politzer has pointed out, the sinus is more forward in diploëtic and slightly cellular bones, which points are borne out by this series. Iwanoff, previously referred to, states that in brachycephalic skulls the right temporal bone is not so well developed as the left and the right lateral sinus is far forwards and outwards. On this question I am not in a position to judge.

The left lateral sinus groove is, however, not infrequently, the larger, for there are twenty-six instances, a proportion much larger than one expected. It is more frequent in males than females.

1. $\frac{11}{12}$ $2\frac{2}{12}$.
2. $\frac{13}{14}$ M. $3\frac{3}{12}$.
3. $\frac{39}{40}$ F. 28.
4. $\frac{45}{46}$ M. 30.
5. $\frac{55}{56}$ M. 33.
6. $\frac{69}{70}$ F. 36.
7. $\frac{71}{72}$ F. 36.
8. $\frac{73}{74}$ M. 37.
9. $\frac{79}{80}$ M. 38.
10. $\frac{85}{86}$ F. 38.
11. $\frac{87}{88}$ M. 39.
12. $\frac{111}{112}$ M. 45.
13. $\frac{115}{116}$ M. 47.
14. $\frac{137}{138}$ F. 50.
15. $\frac{139}{140}$ M. 51.
16. $\frac{153}{154}$ F. 54.
17. $\frac{165}{166}$ M. 57.
18. $\frac{167}{168}$ F. 57.
19. $\frac{173}{174}$ M. 59.
20. $\frac{175}{176}$ M. 59.
21. $\frac{177}{178}$ M. 59.
22. $\frac{185}{186}$ M. 61.
23. $\frac{195}{196}$ F. 62.
24. $\frac{213}{214}$ M. 67.
25. $\frac{217}{218}$ M. 68.
26. $\frac{239}{240}$ F. 89.

Males	16
Females	9
Sex unknown	1
Total	26

The greatest discrepancy in size of the grooves is seen in—

$\frac{111}{112}$ M. 45, where the left is three times the size of the right.
$\frac{147}{148}$ M. 53, where the left is very small.
$\frac{153}{154}$ F. 54, where the right is very small.
$\frac{185}{186}$ M. 61, where the left is very much larger than the right.

Equality in size and forwardness is seen in—

$\frac{41}{42}$ F. 28 Both grooves are large and both bones are mostly diploëtic.

$\frac{107}{108}$ M. 44. Both grooves are large and both bones diploëtic.

$\frac{145}{146}$ M. 52. Both grooves are large, the right bone being cellular and the left partly cellular and partly diploëtic.

The larger sinus is not always the more forward, for in the bilateral diploëtic and slightly cellular types both sinuses may be equally well forward, even though differing in size.

$\frac{101}{102}$ F. 37, where both sinuses are large and equally well forward, the right being somewhat the larger. Both bones are diploëtic.

$\frac{111}{112}$ M. 45, where the right sinus is one third the size of the left and yet both are equally forward. The right bone is almost entirely diploëtic and the left is cellular except for the lower mastoid, which is diploëtic.

Extreme forwardness, the sinus reaching the posterior meatal wall, and being especially large in size, is seen in—

$\frac{109}{110}$ M. 45 on both sides, shutting out the apex of the antrum from the surface, both bones being diploëtic.

$\frac{151}{152}$ F. 54 on the right side, completely shutting out the antrum from the surface, the bone being diploëtic. The left bone is cellular.

$\frac{183}{184}$ M. 60 on the right side, shutting out the apex of a high lying antrum, the bone being diploëtic. The left bone is cellular.

Great forwardness in a cellular bone is seen in—

$\frac{91}{92}$ M. 39 on the right side, with a high lying antrum.

$\frac{285}{286}$ M. 82 on the right side.

The sinus is not always well forward in the diploëtic or slightly cellular types.

$\frac{87}{88}$ M. 28 where both bones are diploëtic and the right sinus is large and well forward and the left is not.

$\frac{43}{44}$ F. 29. Ditto.

$\frac{137}{138}$ F. 50 where both bones are diploëtic and the left sinus is large and well forward and the right is not.

$\frac{193}{194}$ F. 61, where the right bone is diploëtic and the left finely cellular throughout, neither sinus being well forward.

A deep pocket or sulcus in the left lateral sinus groove just before its exit is present in $\frac{115}{116}$ M. 47.

(*b*) *The mastoid emissary vein.*—This vein usually leaves the lateral sinus at a corresponding point on the two sides; although that point may vary immensely, it is in the great majority of cases, below the knee of the vessel, for in one set only does it come off above the knee on both sides, the right vein being the larger and the left rudimentary:

$\frac{95}{96}$ M. 39.

The internal opening in the bone is most frequently in the groove itself, or in its lower edge, but it may be some distance away, the vein running in an open groove before entering the bone, as on both sides of—

$\frac{119}{120}$ M. 48,

or on one side only, as in—

$\frac{175}{176}$ M. 59.

The point of emergence, which is subject to considerable variation in position, is usually symmetrical, but one may be further back than the other, as in—

$\frac{13}{14}$ M. $3\frac{3}{12}$, where the left is in the occipital suture and the right farther forwards.

One side may be higher than the other, one running upwards and the other downwards, as in—

$\frac{147}{148}$ M. 53, where the right runs downwards and the left upwards.

A regular sulcus at the point of emergence is occasionally seen:

On the right side only in—

$\frac{17}{18}$ M. 9, with a diploic vein opening into it above,

and,

$\frac{127}{128}$ M. 49.

$\frac{183}{184}$ F. 61, where the right starts double and emerges single.

The knowledge of the possible variations is important in cases of thrombosis of the lateral sinus when the vein is likely to be implicated and require dealing with.

(v) *The inferior surface*

(*a*) *The sulcus jugularis* is broadly speaking larger and higher lying on the side which has the larger lateral sinus namely the right and this is especially well marked in—

$\frac{35}{36}$ M. 28.
$\frac{103}{104}$ M. 43.
$\frac{121}{122}$ F. 48.
$\frac{147}{148}$ M. 53.
$\frac{211}{212}$ F. 66.

In 32 sets, however, the left sulcus is larger than the right.

1. $\frac{7}{8}$ M. 2.
2. $\frac{11}{12}$ $2\frac{2}{12}$.
3. $\frac{15}{16}$ F. 6.
4. $\frac{39}{40}$ F. 28.
5. $\frac{45}{46}$ M. 30.
6. $\frac{51}{52}$ F. 30.
7. $\frac{55}{56}$ M. 33.
8. $\frac{59}{60}$ F. 33.
9. $\frac{69}{70}$ F. 36.
10. $\frac{71}{72}$ F. 36.
11. $\frac{79}{80}$ M. 38.
12. $\frac{85}{86}$ F. 38.
13. $\frac{87}{88}$ M. 39.
14. $\frac{95}{96}$ M. 39.
15. $\frac{105}{106}$ F. 43.
16. $\frac{111}{112}$ M. 45.
17. $\frac{113}{114}$ M. 45.
18. $\frac{115}{116}$ M. 47.
19. $\frac{127}{128}$ M. 49.
20. $\frac{137}{138}$ F. 50.
21. $\frac{139}{140}$ M. 51.
22. $\frac{153}{154}$ F. 54.
23. $\frac{165}{166}$ M. 57.
24. $\frac{167}{168}$ F. 57.
25. $\frac{173}{174}$ M. 59.
26. $\frac{175}{176}$ M. 59.
27. $\frac{177}{178}$ M. 59.
28. $\frac{185}{186}$ M. 61.
29. $\frac{195}{196}$ F. 62.
30. $\frac{213}{214}$ M. 67.
31. $\frac{217}{218}$ M. 68.
32. $\frac{239}{240}$ F. 89.

Males	18
Females	13
Sex unknown	1

It can be seen that this list includes all the sets in which the left lateral sinus is the larger, but leaves six over:

1. $\frac{15}{16}$ F. 6, where the right lateral sinus is somewhat the larger and the mastoid viens are equal.
2. $\frac{51}{52}$ F. 30, where the bones do not allow of comparison of the lateral sinuses and mastoid veins.

On the left side only in—
$\frac{135}{136}$ F. 62.
On both sides in—
$\frac{57}{58}$ M. 33.

The size of the veins varies enormously; one side may be larger than the other, the larger being frequently associated with the larger lateral sinus, as in—

$\frac{77}{78}$ F. 37, where the right lateral sinus is the larger, the left mastoid vein being rudimentary,

but not always so, for the right lateral sinus may be the larger with the smaller mastoid vein, as in—

$\frac{55}{56}$ M. 28,
$\frac{161}{162}$ M. 56.

The vein may be indistinguishable on both sides, as in—

$\frac{115}{116}$ M. 47,
$\frac{239}{240}$ F. 89;

or it may be very small on both sides, as in—

$\frac{119}{120}$ M. 48,

or indistinguishable on one side and very small in the other, as in—

$\frac{153}{154}$ F. 54, the left being very small and the right indistinguishable.

It may be double at the start and exit on both sides, as in—

$\frac{229}{230}$ F. 76.

It may be double at the start and exit on one side and single at the start and exit on the other, as in—

$\frac{37}{38}$ F. 22, the right side being double.

It may be single at the start and double at the exit on both sides as in—

$\frac{57}{58}$ M. 33.

It may be single at the start and double at the exit on one side and single at the start and exit on the other, as in—

$\frac{71}{72}$ F. 36, where the left is double at the exit.
$\frac{73}{74}$ M. 37, where the left is double at the exit.
$\frac{105}{106}$ F. 43, where the right is double at the exit.
$\frac{215}{216}$ M. 82, where the right is double at the exit.

It may be double at the start and single at the exit on one side and single at both start and exit on the other, as in—

3. $\frac{95}{96}$ M. 39, where the left sulcus is somewhat the larger; both lateral sinuses are large, the right being slightly the larger, but the right mastoid vein coming off above the knee of the sinus is larger than the left, which is rudimentary.

4. $\frac{105}{106}$ F. 43, where the right mastoid vein and lateral sinus are the larger, but the left mastoid vein is rudimentary.

5. $\frac{113}{114}$ M. 45, in which set the bones do not allow of comparison of the lateral sinuses and mastoid veins.

6. $\frac{127}{128}$ M. 49, where both sulci are small, but the left is decidedly larger than the right. The right lateral sinus is the larger, but has a large mastoid vein, while the left mastoid vein is rudimentary.

Nos. 3, 4 and 6 of the above suggest that the size of the sulcus may sometimes depend on the size of the mastoid vein. Other sets also support this:

$\frac{35}{36}$ M. 28,
$\frac{109}{110}$ M. 45,
in both of which sets the right lateral sinus and sulcus are the larger, the left sulcus being out of all proportion smaller than the right, and the left mastoid vein being much larger.

$\frac{219}{220}$ M. 71, where both sulci are large and high lying and both mastoid veins are small.

But against such a suggestion are the following sets, which also show that a large lateral sinus does not always mean a large sulcus:

$\frac{183}{184}$ M. 60, where the right lateral sinus is large and the mastoid vein and sulcus are small, and—

$\frac{203}{204}$ M. 64, where both lateral sinuses are large and both sulci small, the right mastoid vein being rudimentary and the left small.

The position of the sulcus influences the spread of cells inwards as in—

$\frac{63}{64}$ M. 35,
$\frac{103}{104}$ M. 43,
$\frac{139}{140}$ M. 51,
where the smaller sulcus allows of a larger spread of cells under the labyrinth.

High out-pushing sulci are seen in all types of bone.

(*b*) *Marked bulging of cells forming a bulla internally to, and behind, the mastoid process* (*digastric bulla*) is seen in thirteen sets:

1. $\frac{25}{26}$ F. 21 on both sides, more marked on the right
2. $\frac{29}{30}$ M. 24 on the right side only.
3. $\frac{39}{40}$ F. 28 on both sides.
4. $\frac{103}{104}$ M. 43 on the right side only.
5. $\frac{131}{132}$ F. 49 on both sides.
6. $\frac{139}{140}$ M. 51 on both sides, more marked on the left.
7. $\frac{141}{142}$ M. 51 on both sides.
8. $\frac{145}{146}$ M. 52 on the right side only.
9. $\frac{195}{196}$ F. 62 on both sides, more marked on the left, where it runs up to the sulcus jugularis.
10. $\frac{207}{208}$ F. 64 on both sides, more marked on the left.
11. $\frac{209}{210}$ M. 66 on both sides.
12. $\frac{235}{236}$ M. 82 on the left side only.
13. $\frac{237}{238}$ F. 86 on both sides, more marked on the right.

In all these pus is liable to perforate into the neck internally to the mastoid process.

(*c*) *Specially large idgastric fossæ* are seen in four sets:

1. $\frac{41}{42}$ F. 28, the left being the larger.
2. $\frac{49}{50}$ M. 30 on both sides.
3. $\frac{53}{54}$ M. 33 on the right side, the left not allowing of examination.
4. $\frac{59}{60}$ F. 33 on the right side, the left not allowing of examination.

2. THE INTERIOR

(*i*) *Types*

Classification of the types is founded on the conditions of the outer antral wall and mastoid process. In referring to the mastoid process the terms "upper and lower mastoid" are used, the "upper mastoid" being the bone below the level of the apex of the antrum down to the upper level of the projecting part or "lower mastoid."

This division is justified by the totally different surgical anatomical relationships.

On this basis it is found that of the 120 sets 82 are symmetrical and 38 asymmetrical. In many of the asymmetrical bones there is a tendency to symmetry.

It may be here stated that there are very few cellular bones without remains of diploë, either as a small mass at the extreme tip of the mastoid process, or as a rim round the tip.

Asymmetry as to extension of cells is indicated with each specimen.

Symmetrical

(*a*) Dense outer antral wall lined internally with cells which can be seen at the eighth month of fœtal life, and therefore called fœtal cells, with a dense layer of bone between the antrum and a diploëtic mastoid. *Diploetic infantile type*........ 24

For my description of this type and its surgical importance reference can be made to the Transactions of the Eighth International Congress of Otology held in Buda-Pesth; in that paper, the importance of the type in conducing to intra-cranial complications in acute infection of the antrum and to chronic middle-ear suppuration, and its sequelæ, apart from tuberculosis, was pointed out.

1.	$\frac{1}{2}$	M.	$\frac{9}{12}$.
2.	$\frac{3}{4}$	F.	$1\frac{7}{12}$.
3.	$\frac{5}{6}$	?	$1\frac{9}{12}$.
4.	$\frac{7}{8}$	M.	2.
5.	$\frac{11}{12}$	?	$2\frac{2}{12}$.
6.	$\frac{13}{14}$	M.	$4\frac{8}{12}$.
7.	$\frac{15}{16}$	F.	6.
8.	$\frac{87}{88}$	M.	28.
9.	$\frac{41}{42}$	F.	28.
10.	$\frac{45}{46}$	M.	30.
11.	$\frac{71}{72}$	F.	36.
12.	$\frac{95}{96}$	M.	39.
13.	$\frac{101}{102}$	F.	42.
14.	$\frac{107}{108}$	M.	44.
15.	$\frac{109}{110}$	M.	45.
16.	$\frac{187}{188}$	F.	50.
17.	$\frac{149}{150}$	F.	53.
18.	$\frac{153}{154}$	F.	54.
19.	$\frac{155}{156}$	F.	55.
20.	$\frac{169}{170}$	M.	58.
21.	$\frac{181}{182}$	M.	60.
22.	$\frac{213}{214}$	M.	67.
23.	$\frac{217}{218}$	M.	68.
24.	$\frac{231}{232}$	F.	77.

Males..............................	12
Females............................	10
Sex unknown........................	2

Children up to 6
years of age.......................... 7
Adults..................................17

(*b*) Dense outer antral wall and very few cells in the upper mastoid. Diploētic lower mastoid................ 1

1. $\frac{31}{32}$ M. 24.

(*c*) Dense outer antral wall. Cellular upper mastoid. Diploētic lower mastoid.................................... 5

1. $\frac{23}{24}$ M. 20.
2. $\frac{27}{28}$ F. 22.
3. $\frac{49}{50}$ M. 30.
4. $\frac{161}{162}$ M. 56.
5. $\frac{201}{202}$ M. 64.

(*d*) Dense outer antral wall. Upper and lower mastoid cellular.................................... 1

$\frac{191}{192}$ F. 61. More diploē at the tip of the left mastoid than on the right.

(*e*) Diploētic outer antral wall. A few cells in upper mastoid. Lower mastoid diploētic........................ 1

$\frac{203}{204}$ M. 64.

(*f*) Cellular outer antral wall and upper mastoid. Lower mastoid diploētic...................................12

1. $\frac{9}{10}$ F. 2.
2. $\frac{47}{48}$ M. 30.
3. $\frac{65}{66}$ F. 35.
4. $\frac{99}{100}$ M. 42.
5. $\frac{119}{120}$ M. 48.
6. $\frac{143}{144}$ M. 51.
7. $\frac{147}{148}$ M. 53.
8. $\frac{165}{166}$ M. 57.
9. $\frac{171}{172}$ M. 58.
10. $\frac{183}{184}$ M. 60.
11. $\frac{221}{222}$ M. 72.
12. $\frac{223}{224}$ M. 72.

(*g*) Cellular outer antral wall and entire mastoid, some having more dipoē at the tip than the others, and some more one side than the other...................................38

1. $\frac{21}{22}$ F. 16.
2. $\frac{25}{26}$ F. 21. Digastric bulla on both sides, more marked on the right.
3. $\frac{29}{30}$ M. 24. Digastric bulla on the right side only.
4. $\frac{33}{34}$ F. 25.
5. $\frac{39}{40}$ F. 28. Digastric bulla on both sides.
6. $\frac{51}{52}$ F. 30.
7. $\frac{61}{62}$ M. 34.

8. $\frac{63}{64}$ M. 35. The occipital diploë is invaded by cells on the right side and not on the left.

9. $\frac{67}{68}$ M. 36.

10. $\frac{69}{70}$ F. 36.

11. $\frac{79}{80}$ M. 38.

12. $\frac{81}{82}$ M. 38. Extension inwards, behind the middle ear under the labyrinth and invading the lower part of the internal diploë on both sides.

13. $\frac{83}{84}$ F. 38.

14. $\frac{85}{86}$ F. 38.

15. $\frac{89}{90}$ M. 39. The cells on the left side only extend inwards behind the middle ear and under the labyrinth.

16. $\frac{91}{92}$ M. 39.

17. $\frac{103}{104}$ M. 43. The cells extending inwards under the labyrinth are larger on the left side where the sulcus jugularis is not so high.
Digastric bulla on the right side only

18. $\frac{117}{118}$ M. 48.

19. $\frac{123}{124}$ F. 48. Mastoid cortex dense and thick on the right side and thin on the left.

20. $\frac{131}{132}$ F. 49. Digastric bulla on both sides.

21. $\frac{133}{134}$ M. 50.

22. $\frac{135}{136}$ F. 50.

23. $\frac{139}{140}$ M. 51. The cells extend over the meatus into the zygoma on both sides. Extension inwards behind the middle ear under the labyrinth and invading the lower part of the internal diploë on both sides, but more on the right, on which side the sulcus jugularis is not so high. Digastric bulla on both sides, more marked on the left.

24. $\frac{141}{142}$ M. 51. Digastric bulla on both sides, more marked on the right.

25. $\frac{159}{160}$ F. 55.

26. $\frac{167}{168}$ F. 57.
27. $\frac{179}{180}$ M. 59.
28. $\frac{185}{186}$ M. 61.
29. $\frac{189}{190}$ F. 61.
30. $\frac{195}{196}$ F. 62. Digastric bulla on both sides, more marked on the left, where it runs inwards to the sulcus jugularis.
31. $\frac{197}{198}$ F. 62.
32. $\frac{207}{208}$ F. 64. Digastric bulla on both sides, more marked on the left.
33. $\frac{209}{210}$ M. 66. Digastric bulla on both sides.
34. $\frac{211}{212}$ F. 66. Cells extending up into the squama on the right side only.
35. $\frac{219}{220}$ M. 71.
36. $\frac{225}{226}$ F. 72.
37. $\frac{235}{236}$ M. 82. Digastric bulla on the left side only.
38. $\frac{237}{238}$ F. 86. Digastric bulla on both sides, more marked on the right.

Males	18	
Females	20	
Total	38	
Total symmetrical		82

Asymmetrical

In many of the asymmetrical sets it will be seen that there is a decided tendency to symmetry.

(*a*) Diploëtic infantile type on one side (*a*).
Fine cells in the outer antral wall and diploëtic mastoid on the other; a rare condition (*b*) 2

1. (*a*) $\frac{43}{44}$ F. 29.
2. (*b*) $\frac{199}{200}$ F. 63.

In these sets suppuration is likely to perforate the outer antral wall on the left side.

(*b*) Diploëtic infantile type on one side (*a*). Dense outer antral wall with a few cells in the upper part of a diploëtic mastoid on the other (*b*) 5

1. $\frac{(b)\,19}{(a)\,20}$ M. 11.
2. $\frac{(b)\,57}{(a)\,58}$ M. 33.

3. $\frac{(a)\,77}{(b)\,78}$ F. 37.
4. $\frac{(a)\,127}{(b)\,128}$ M. 49.
5. $\frac{(b)\,187}{(a)\,188}$ M. 61.

(*c*) Diploëtic infantile type, on one side (*a*). Dense outer antral wall, cellular upper mastoid and diploëtic lower mastoïd, on the other (*b*)..........................3

1. $\frac{(a)\,35}{(b)\,36}$ M. 28.
2. $\frac{(b)\,115}{(a)\,116}$ M. 47.
3. $\frac{(a)\,121}{(b)\,122}$ F. 48.

(*d*) Diploëtic infantile type, on one side (*a*). Cellular outer antral wall and upper mastoid and diploëtic lower mastoid, on the other (*b*)..............................5

1. $\frac{(a)\,93}{(b)\,94}$ M. 39.
2. $\frac{(b)\,129}{(a)\,130}$ M. 49.
3. $\frac{(a)\,151}{(b)\,152}$ F. 54.
4. $\frac{(b)\,173}{(a)\,174}$ M. 59.
5. $\frac{(a)\,233}{(b)\,234}$ F. 81.

(*e*) Diploëtic infantile type, on one side (*a*). Cellular outer antral wall and entire mastoid, on the other (*b*)........5

1. $\frac{(a)\,97}{(b)\,98}$ F. 41.
2. $\frac{(b)\,105}{(a)\,106}$ F. 43. The cells in the right are small and surrounded by dense bone; diploë at the tip of the mastoid.
3. $\frac{(b)\,163}{(a)\,164}$ F. 56.
4. $\frac{(a)\,193}{(b)\,194}$ F. 61.
5. $\frac{(a)\,227}{(b)\,228}$ F. 73. Densely diploëtic on the right.

(*c*), (*d*), and (*e*) explain why, in acute bilateral antral infection, a mastoid abscess may result on one side and a chronic discharge without a mastoid abscess on the other.

Those specimens which have only a dense outer wall and a very few cells in the upper part of a diploëtic mastoid have the same surgical importance with regard to the suppuration as the pure diploëtic type, and the same applies to those bones in which the entire mastoid is composed of cells surrounded by a thick layer of dense bone.

There are, therefore, twenty sets in which the diploëtic infantile type is seen on one side only. On the right side in twelve, and on the left side in eight.

Nine are in males and eleven in females.

It is interesting to note that in the whole series of a hundred and twenty individuals the type is seen in forty-four; both sides in twenty-four, and on one side in twenty.

(*f*) Dense outer antral wall, cellular upper mastoid, and diploëtic lower mastoid, on one side (*a*).

Dense outer antral wall and entirely cellular mastoid, on the other (*b*)..3

1. $\frac{(a)\,113}{(b)\,114}$ M. 45.
2. $\frac{(a)\,205}{(b)\,206}$ F. 64.
3. $\frac{(a)\,239}{(b)\,240}$ F. 89.

(*g*) Dense outer antral wall, cellular upper mastoid and diploëtic lower mastoid, on one side (*a*).

Cellular outer antral wall and upper mastoid and diploëtic lower mastoid, on the other (*b*)..................3

1. $\frac{(a)\,73}{(b)\,74}$ M. 37. Very few cells in the upper mastoid on the right side.
2. $\frac{(a)\,111}{(b)\,112}$ M. 45. Very few cells in the upper mastoid on the right side
3. $\frac{(b)\,157}{(a)\,158}$ F. 55.

(*h*) Dense outer antral wall, cellular upper mastoid and diploëtic lower mastoid, on one side (*a*).

Cellular outer antral wall and entire mastoid on the other. (*b*)..2

1. $\frac{(a)\,125}{(b)\,126}$ M. 49.
2. $\frac{(a)\,175}{(b)\,176}$ M. 59.

(*i*) Dense outer antral wall and entire mastoid cellular, on one side (*a*).

Cellular outer antral wall and entire mastoid, on the other (*b*) 1

1. $\frac{(b)\,87}{(a)\,88}$ M. 39.

(*j*) Cellular outer antral wall and upper mastoid, lower mastoid diploëtic, on one side (*a*).

Cellular outer antral wall and entire mastoid, on other (*b*)..6

1. $\frac{(a)\,17}{(b)\,18}$ M. 9.
2. $\frac{(b)\,53}{(a)\,54}$ M. 33.
3. $\frac{(b)\,145}{(a)\,146}$ M. 52. On the right side, the cells extend inwards from the mastoid, forming a digastric bulla.

4. $\frac{(b)\,177}{(a)\,176}$ M. 59.
5. $\frac{(b)\,215}{(a)\,216}$ M. 68.
6. $\frac{(b)\,229}{(a)\,230}$ F. 76.

These sets show that in double mastoid infection the lower mastoid is not necessarily affected on both sides.

Diploë in the outer antral wall is very unusual; it is present on one side only in the three following sets. Reference has already been made to $\frac{203}{204}$ M. 64, where diploë is present in the outer antral wall on both sides.

(*k*) Diploëtic outer antral wall and entire mastoid, on side (*a*).

Dense outer antral wall with a few cells just below the apex of the antrum, the remainder of the mastoid being diploëtic, on the other (*b*) 2

1. $\frac{(b)\,55}{(a)\,56}$ M. 33.

The left bone in this set is a rare and interesting one. The section shows the diploë in the zygomatic squamous and petrous elements, separated from one another by distinct partitions of compact bone, and the squamous diploë forming the outer antral wall, which is half an inch in thickness, is separated from the cells which always line the outer antral wall (fœtal cells) by another thin layer of compact bone. On the right side the dense outer antral wall is formed by thickening of the compact bony partition between the zygomatic and squamous masses of diploë.

This type of bone is responsible for those cases of infection from the antrum which run an osteomyelitic course.

2. $\frac{(a)\,59}{(b)\,60}$ F. 33.

The right bone of this set is very like the left one of the previous set, but the layer of dipoë in the outer antral wall is much thinner and the compact layer between the diploë and the fœtal cells is much thicker.

(*l*) *3.* Diploëtic outer antral wall, cellular upper mastoid and diploëtic lower mastoid, on one side (*a*)

Dense outer antral wall with cells in the base of the upper mastoid, the remainder of which is diploëtic, on the other (*b*) 1

1. $\frac{(b)75}{(a)76}$ M. 37.

The left bone shows a thin outer layer of diploë formed by the zygomatic squamous and mastoid elements with distinct thin separating layers of compact bone. The zygomatic element forms the outer antral wall, the antrum being high lying.

Total asymmetrical....................................38

(ii) *The size and shape of the antrum* are subject to very slight variation on the two sides; a difference, and that a small one, is only seen in two sets—

1. $\frac{107}{108}$ M. 44,
2. $\frac{149}{150}$ F. 53,

In both of which sets the left antrum is smaller than the right, both bones being of the diploëtic infantile type.

(iii) *High lying antrum* in relation to the posterior zygomatic line is seen in nineteen sets:

1. $\frac{37}{38}$ M. 28. Bilateral diploëtic infantile type.
2. $\frac{39}{40}$ F. 28. Higher on the right side.
3. $\frac{53}{54}$ M. 33.
4. $\frac{61}{62}$ M. 34.
5. $\frac{73}{74}$ M. 37. Higher on the right side, where the bone is nearly of the pure diploëtic infantile type for there is only one small cell in the mastoid diploë.
6. $\frac{75}{76}$ M. 37.
7. $\frac{87}{88}$ M. 39.
8. $\frac{91}{92}$ M. 39.
9. $\frac{103}{104}$ M. 43.
10. $\frac{111}{112}$ M. 45.
11. $\frac{139}{140}$ M. 51.
12. $\frac{147}{148}$ M. 53. On the right side only.
13. $\frac{177}{178}$ M. 59. Higher on the right side.
14. $\frac{183}{184}$ M. 60.
15. $\frac{185}{186}$ M. 61. On the right side only.
16. $\frac{195}{196}$ F. 62. Higher on the right side.
17. $\frac{213}{214}$ M. 67. Bilateral diploëtic infantile type.
18. $\frac{221}{222}$ M. 72.
19. $\frac{223}{224}$ M. 72.

It is in the great majority of cases symmetrical, for in two sets only is the antrum high lying on one side and not on the other:

$\frac{147}{148}$ M. 53,
$\frac{165}{166}$ M. 61,

Where the right in each instance is the high lying one.

In four sets one antrum is higher than the other, and the higher one is, in all, on the right side:

1. $\frac{39}{40}$ F. 28.
2. $\frac{73}{74}$ M. 37.
3. $\frac{177}{178}$ M. 59
4. $\frac{195}{196}$ F. 62.

It is more common in males than in females, for seventeen are males and only two are females.

It is most commonly seen in the cellular bones, for in two sets only is it present in the diploëtic infantile type, and in them on both sides

1. $\frac{87}{88}$ M. 28.
2. $\frac{213}{214}$ M. 67.

The antrum may be high lying and yet the middle fossa may dip down, partly shutting off the cavity from the surface as, on the right side, in

$\frac{111}{112}$ M. 45.

There is no reliable external guide to a high lying antrum, except that a posterior zygomatic line which slopes downwards and backwards should put one on one's guard.

(iv) *The sinus tympani* or internal pyramidal recess cannot be examined in all specimens, but the sets,

$\frac{171}{172}$ M. 58,
$\frac{203}{204}$ M. 64,

deserve careful study, for an extraordinarily large cavity is seen on both sides in each. This cavity may be infected in suppuration and responsible for chronic discharge with or without antral infection and for the continuation of discharge after a radical operation has been performed. Unless the cavities are as large as these, it is practically impossible to deal with them without injury to the facial nerve; but if they are large they can be opened immediately below the external semi-circular canal and behind the level of the descending part of the facial nerve, cleaned of contents and laid well open into the bony wound, as in a case of a boy, aged 9 years, described by myself in the 'Transactions of the Royal Society of Medicine, Otological Section,' vol. iv, No. 4, p. 54.

It is interesting to note that Mr. G. J. Jenkins considers this cavity as the upper end of the first branchial cleft.

The diagnosis of disease in this cavity cannot be dealt with in this paper.

(v) *Malformation of the bony external semi-circular canal* is seen in 191, the right bone of a woman, aged 61 years. The canal is represented by a single large cavity opening widely into the outer wall of the vestibule. A small tubercle on the upper part of the sloping posterior wall of the cavity represents the only attempt at the formation of the normal partition.

The canals on the left side are normal.

B. In Disease

(i) *Injuries*

Fracture through the bone is seen in three sets only.

1. *23.* The right bone of a male, aged 20 years. The fracture passes through the middle ear and posterior part of the labyrinth dividing the facial nerve.

2. *180.* The left bone of a male, aged 59 years. The fracture passes through the middle ear, meatus, and mastoid cells just externally to the external semi-circular canal. The facial nerve is not divided.

3. *184.* The left bone of a male, age 60 years The fracture passes right across the bone just externally to the external semi-circular canal dividing the facial nerve in its descending course.

(ii) *Changes in bone*

(*a*) *Exostoses in the meatus* on both sides more marked on the right in—

$\frac{165}{166}$ M. 57.

(*b*) *A pedunculated exostosis* is present on the inner wall of a large cell *in the lower mastoid* of the right bone of—

179. M. 59.

(*c*) *Hyperostosis of the tympanic plate* on both sides, in—

$\frac{215}{216}$ M. 68.

(*d*) *Thinning of the tympanic plate*, due to the presence of masses of epithelium, keratosis obturans, is seen on both sides of—

$\frac{85}{86}$ F 38,

being more marked on the left. Evidence of past suppuration is seen in both membranes.

(*e*) *Bony excrescences on the inner aspect of the bone* are seen in five sets, all associated with mental trouble:

1 $\frac{49}{50}$ F 30. A general paralytic. An exostosis is seen on the lower edge of the lateral sinus groove on the right side only.

2. $\frac{65}{66}$ F. 35. An epileptic dement. The excrescences are present on the inner surface of the squama on both sides, but more marked on the left. There is also great thickening of the parietal bone.

3. $\frac{75}{76}$ M. 37. An epileptic dement. An esostosis is present on the lower edge of the lateral sinus groove, on the left side only. The markings of the cerebral convolutions are very pronounced on both sides.

4. $\frac{131}{132}$ F 49. A melancholic. The excrescences are seen on the inner aspect of the squama, on the left side especially.

5. $\frac{237}{238}$ F 86 A senile dement. The same as the preceding specimen.

(*f*) *Great thickening of the skull with corrugations and deep groovings* due to osteitis deformans, is seen on both sides in—

1. $\frac{229}{220}$ F. 76.

The thickening is seen in the parietal bones, the temporal bone escaping altogether, but the inner aspect of the squama is corrugated and deeply grooved for vessels.

(*g*) *Extreme dilatation, with thinning of the left internal* *auditory meatus*, due to the presence of a cerebellar tumour, is seen in 52, the left bone of a female, aged 30.

(*h*) *Fixation of the stapes* is seen in—

$\frac{71}{72}$ F. 36.

This patient had been getting deaf since childhood. For many years, about eight, she had been insane with subacute mania. Death was due to pulmonary and intestinal tuberculosis. She had very marked hallucinations of hearing, hearing voices and chattering to imaginary persons. She also gave a rambling and confused account of a medical student whom she loved and who had promised to marry her after he had operated on her for deafness. She spoke in a slow wailing monotone.

Her father had been deaf since boyhood and at one time heard better in a noise.

Both bones are of the diploëtic infantile type, and both membranes are intact.

Right bone.—Outer vestibular wall.

(*i*) From the vestibular side. The posterior half of the base of the stapes appears thickened and pushed well through the oval window and fixed, while the anterior is not thickened and appears a little tilted out from the window as if the stapedius muscle had exerted a permanent effect. The anterior half of the upper lip of the oval window is much thickened.

(*ii*) From the tympanic side. The inner half of the crura of the stapes are buried in new tissue, the bone being absolutely fixed.

Left bone.—Outer vestibular wall.

(*i*) From the vestibular side. Very similar to the right side.

(*ii*) From the tympanic side. The crura of the stapes (fractured accidentally) are not so buried as on the right side, but the base is firmly fixed.

It is interesting to note that both bones are of the diploëtic infantile type.

(*iii*) *Evidence of suppuration is seen in thirteen sets.* The type of bone on both sides in each set is described.

(*a*) Evidence in the membrane without sign of antral infection.

1. $\frac{75}{76}$ M. 37. *Right bone.*—Calcareous deposit in the anterior and posterior segments. No sign of antral infection. Dense outer antral wall with a few cells in the upper part of a diploëtic mastoid.

Left bone.—Crescentic calcareous deposit, with a dry perforation in the anterior inferior segment.

Ossicles intact. No sign of antral infection. Diploëtic and cellular outer antral wall, cellular upper mastoid, diploëtic lower mastoid.

In the writer's opinion calcareous deposits are always the result of a pyogenic infection.

2. $\frac{85}{86}$ F. 38. *Right bone.*—Cicatrix in the posterior segment.

Tympanic plate thinned by the presence in the meatus of layers of epithelium (keratosis obturans). No sign of antral infection. Cellular outer antral wall and mastoid, the tip of which is diploëtic.

Left one.—Dry perforation in the posterior superior segment.

Ossicles intact. Meatus markedly dilated, the tympanic plate being very much thinned by keratosis obturans. No sign of antral infection.

Cellular outer antral wall and entire mastoid.

These two sets point to the fact that the antrum does not always share in infection of the lower middle ear, for if the cavity was ever infected in them, it is difficult to understand why an acute mastoid abscess did not result. The cavity is more likely to be primarily implicated in virulent infection due to scarlet fever, measles, and influenza. In specimens like these, however, a fresh or the old infection may at any time spread to the antrum and mastoid cells, producing acute mastoid signs and symptoms.

3. $\frac{181}{182}$ M. 60.—*Right bone,*—Membrane was intact, but injured on section. No sign of infection.
Diploëtic infantile type.
Left bone.—Large dry inferior kidney shaped perforation. Ossicles intact. No sign of antral infection.
Diploëtic infantile type.

In this left bone it would have been impossible for a mastoid abscess to form if the antrum had been also infected.

(*b*) Evidence in the membrane only on one side and in the antrum as well on the other.

1. $\frac{97}{98}$ F. 41. *Right bone.*—Dry perforation in the inferior segment. Ossicles intact. No sign of antral infection.
Dense outer antral wall, entire mastoid cellular.
Left bone.—Anterior and posterior segments cicatricial.
No perforation. Ossicles intact. The antral walls are smoothed out as if the cavity had at one time been infected. No cholesteatoma. Diploëtic infantile type.

In this right bone, infection of the antrum must have led to mastoid disease, this again pointing to the fact that the antrum does not always share in infection with the lower middle ear. In the left, the signs of infection of the antrum seem to be clear and yet healing had occurred, and if mastoid cells had been present mastoid disease would have certainly resulted.

2. $\frac{119}{120}$ M. 48. *Right bone.*—Dry inferior perforation. Ossicles intact. No sign of antral infection.
Cellular outer antral wall and mastoid, the tip of which is diploëtic.
Left bone.—Posterior inferior perforation. Ossicles intact.
The lining membrane of the antrum and mastoid cells appears thickened.
Cellular outer antral wall and mastoid, the cells being surrounded by dense bone.

In this right bone again if the antrum had shared in the infection of the lower middle ear mastoid disease must have resulted.

In the cellular left bone, chronic discharge had been present, but, as previously stated, a bone with mastoid cells surrounded by dense bone is as conducive to a chronic middle-ear discharge as is the pure diploëtic infantile type.

(*c*) Evidence in the middle ear and antrum on bothsides.

1. $\frac{95}{96}$ M. 39. *Right bone.*—Pus in the middle ear. Perforation in Shrapnell's membrane. Loss of the incus and head of the malleus. Erosion of the outer attic wall. Stapes intact Cholesteatoma in the attic and filling the antrum. Smoothing out of the antral walls.

Diploëtic infantile type.

Left bone.—Pathological conditions exactly similar to those on the right side, except that there are calcareous deposits in the anterior and inferior segments of the membrane.

Diploëtic infantile type.

This set demonstrates how symmetry of anatomical type produces symmetrical pathological conditions.

(*d*) Evidence in the middle ear and antrum on one side, the other side being normal.

1. $\frac{31}{32}$ M. 24. *Right bone.*—Normal. Membrane intact. Dense outer antral wall; a few cells are present in the upper part of a diploëtic mastoid.

Left bone.—The radical operation has been performed and the middle fossa opened.

Death occurred from cerebellar abscess; the pathway of infection is unknown.

Diploëtic infantile type.

This left bone illustrates the fact that the diploëtic or infantile type is conducive not only to chronic suppuration, but to its more dangerous sequelæ.

2. $\frac{101}{102}$ F. 42. *Right bone.*—Normal. Membrane intact.

Diploëtic infantile type.

Left bone.—Perforation in the posterior segment. Ossicles intact. No discharge. The inner and posterior antral walls are smoothed out.

Diploëtic infantile type.

It is interesting to note that healing had taken place without operation on the antrum.

3. $\frac{115}{116}$ M. 47. *Right bone.*—Normal. Membrane intact. Dense outer antral wall; cellular mastoid with a diploëtic tip.

Left bone.—Large loss of the membrana tensa. Loss of the articular process of the incus. Thickening of the antral lining membrane.

Diploëtic infantile type.

4. $\frac{137}{138}$ F. 50. *Right bone.*—Normal. Membrane intact.

Diploëtic infantile type.

Left bone.—Pus was present in the middle ear and antrum, which is small. Large loss of the membrana tensa.

Ossicles intact.

Diploëtic infantile type.

In this left bone, if simple treatment did not produce a cure, the trouble would probably have been amenable to an incomplete mastoid operation.

5. $\frac{153}{154}$ F. 54. *Right bone.*—Normal. Membrane was intact, but has split.

Diploëtic infantile type.

Left bone.—Pus in the middle ear and antrum. Perforation in Shrapnell's membrane and posterior segment. Loss of the head of the malleus, incus, and crura of the stapes. Erosion of the outer attic wall. Niche for the oval window filled by new tissue. All the walls of the antrum are smoothed out, the fœtal cells being completely destroyed.

There was no cholesteatoma.

Diploëtic infantile type.

6. $\frac{127}{128}$ M. 49. *Right bone.*—Pus in the middle ear and antrum.

Complete loss of the membrane, malleus, incus and crura of the stapes, the base of which is intact, as can be seen from the vestibular side. The oval window niche is filled with new tissue. Marked erosion of the outer attic and posterior superior deep meatal walls; the inner and posterior antral walls are smoothed out. The mass of fœtal cells has been converted into a mass of dense bone, narrowing the aditus and antral cavity; this localised osteo-sclerotic condition can be easily distinguished from the normally dense outer antral wall. There was no cholesteatoma.

Diploëtic infantile type.

Left bone.—Normal. Membrane intact. The outer antral wall is thick and dense and the mastoid diploëtic, with the exception of a few small cells running downwards from the apex of the antrum.

7. $\frac{231}{232}$ F. 77. *Right bone.*—Pus in the middle ear and antrum.

Perforation was present in Shrapnell's membrane, but details were destroyed accidently on section. Erosion of the outer attic wall. The aditus is much narrowed by thickening of the free border of the squama of the petro-squamosal junction. The antral walls are smoothed out, the fœtal cells being completely destroyed up to the normal dense outer wall. The roof of the antrum is much thickened by osteo-sclerosis.

There was no cholesteatoma.

Diploëtic infantile type.

Left bone.—Normal; membrane intact.

Diploëtic infantile type.

This is the oldest specimen of chronic middle-ear suppuration which I possess. The woman died of senile mania, broncho-pneumonia, and heart failure.

(*e*) The two last sets have been placed together, for the affected bones show that a certain amount of osteo-sclerosis may take place in the walls of the antrum as a result of chronic suppuration, but it can always be distinguished from the normal dense outer antral wall, so characteristic of the normal diploëtic infantile type.

All the bones affected with chronic suppuration with antral implication are of the diploëtic infantile type, with the exception of *120* already described in which there are small mastoid cells surrounded by dense bone, a condition having the same surgical significance as the pure diploëtic infantile type, and, as may be seen in $\frac{101}{102}$F. 42, $\frac{137}{138}$ F. 50, $\frac{153}{154}$ F. 54, $\frac{231}{232}$ F. 77, suppuration was present in the middle-ear tract, including the antrum, on one side only, the other side being normal, and yet the diploëtic infantile type is present on *both* sides, thus adding emphasis to the fact that the conditions present in that type, viz. a dense outer antral wall, lined internally by the fœtal cells and a dense layer of bone separating the antrum from the diploëtic mastoid process, are factors in producing chronic middle-ear discharge, and the density is not a result.

To those who hold that the dense outer antral wall is the result of chronic suppuration, it can be pointed that the condition is present in this series without any signs of suppuration, on both sides in 18, and on one side in 16.

It can be confidently expected that in the future X-ray photography will be of the greatest value in the diagnosis and treatment of suppurative disease.

PHOTOGRAPHS ILLUSTRATING ARTHUR CHEATLE'S PAPER

ON

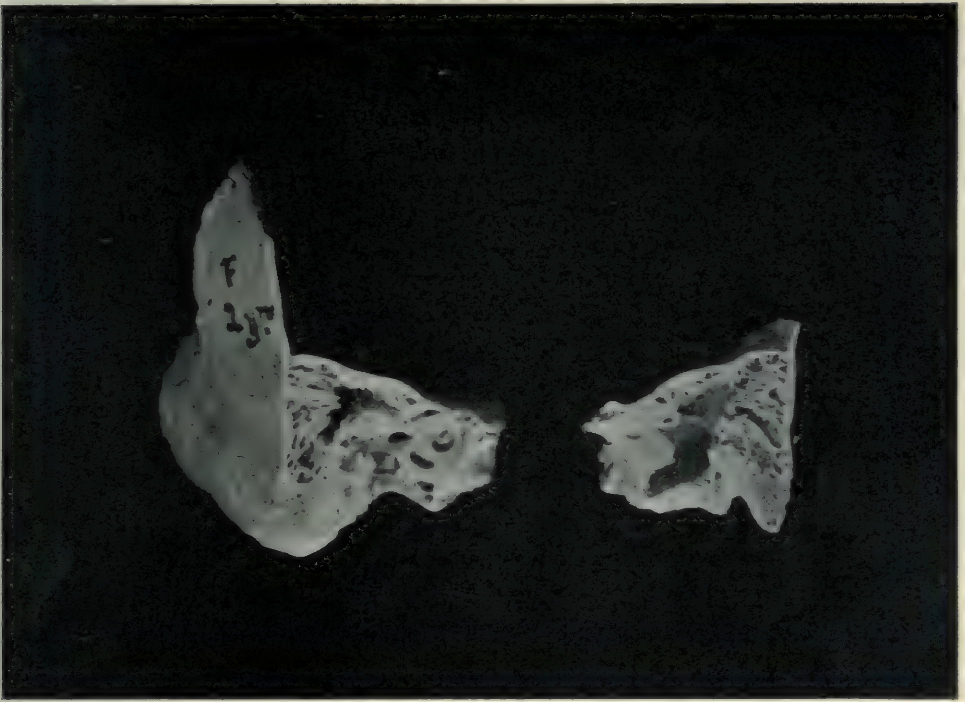
THE REPORT OF AN EXAMINATION OF BOTH TEMPORAL BONES FROM A HUNDRED AND TWENTY INDIVIDUALS IN REFERENCE TO THE QUESTION OF SYMMETRY IN HEALTH AND DISEASE

Presented at the Ninth International Otological Congress,
Boston, 1912.

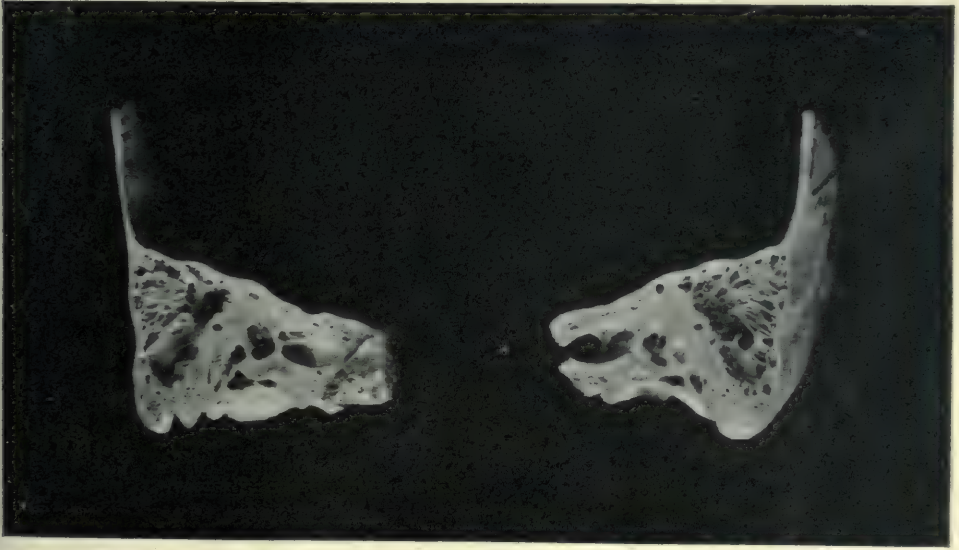
The Numbers of the Figures Have No Relation to Those Given in the Text.

No. 1.—FEMALE, AGED 2 YEARS.

Symmetrical.



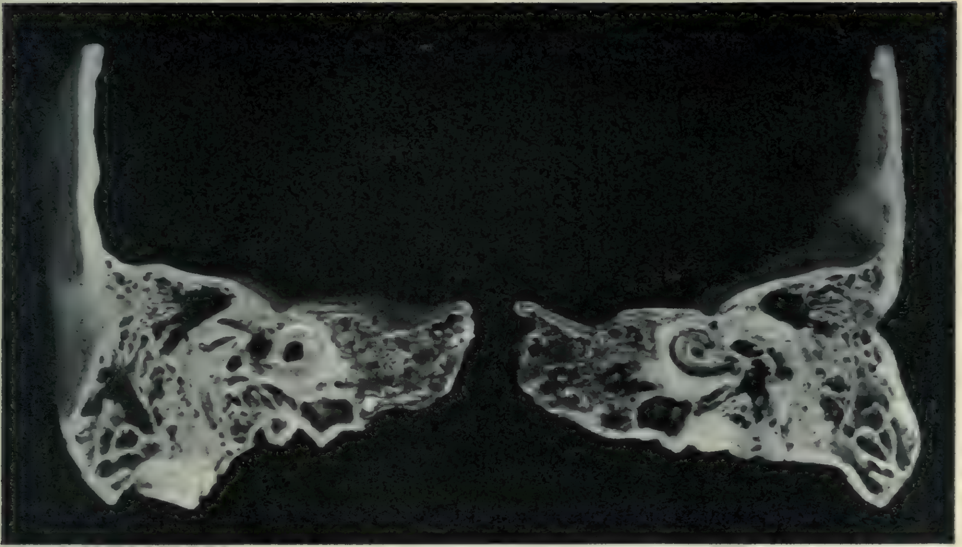
Right.—Long, narrow antrum. Diploe at the tip of the mastoid. Cells from the floor of the middle ear run under the first turn of the cochlea.



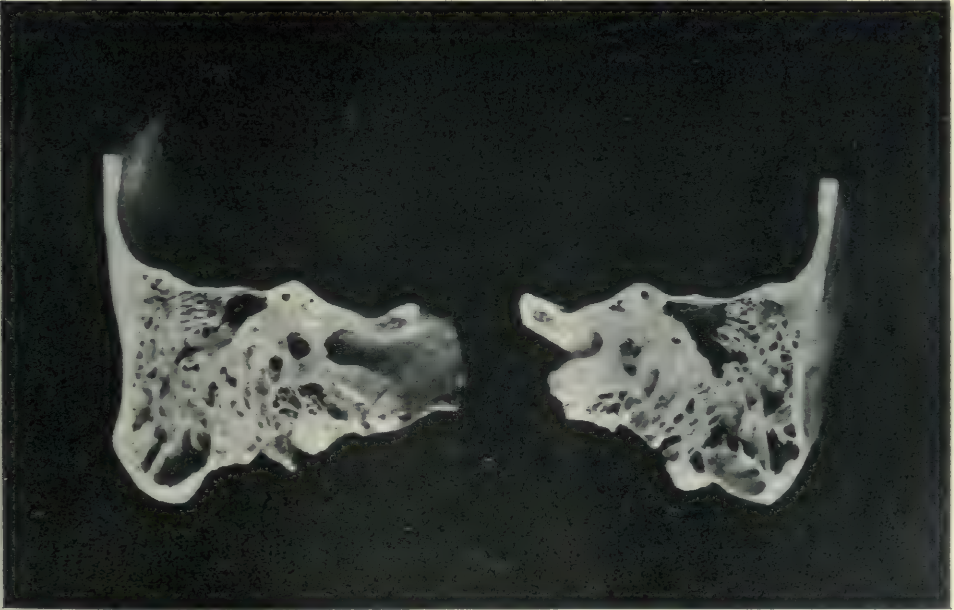
Left.—Symmetrical with the right. The section has been made further back than on the right side.

No. 2.—FEMALE, AGED 16 YEARS.

Symmetrical.



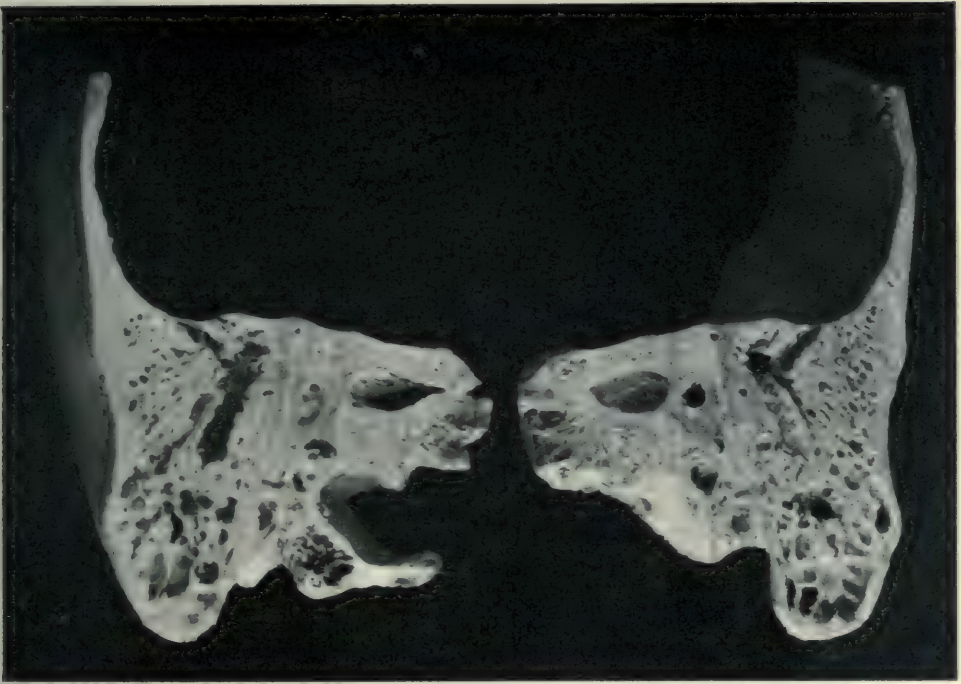
Right.—Outer antral wall and entire mastoid cellular. Cells from the floor of the middle ear pass under the cochlea, reaching and slightly invading the internal diploetic mass.



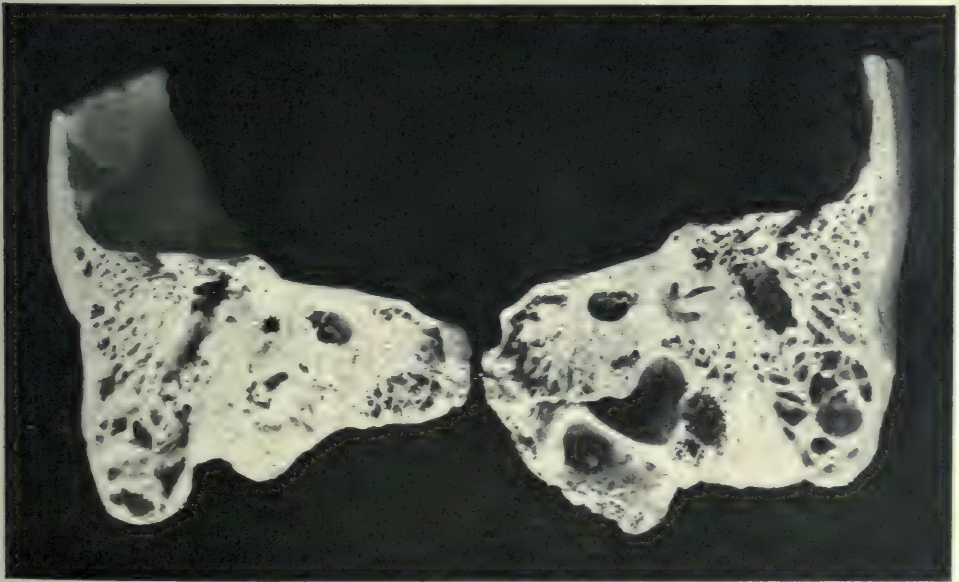
Left.—Symmetrical with the right.

No. 3.—FEMALE, AGED 49 YEARS.

Symmetrical.



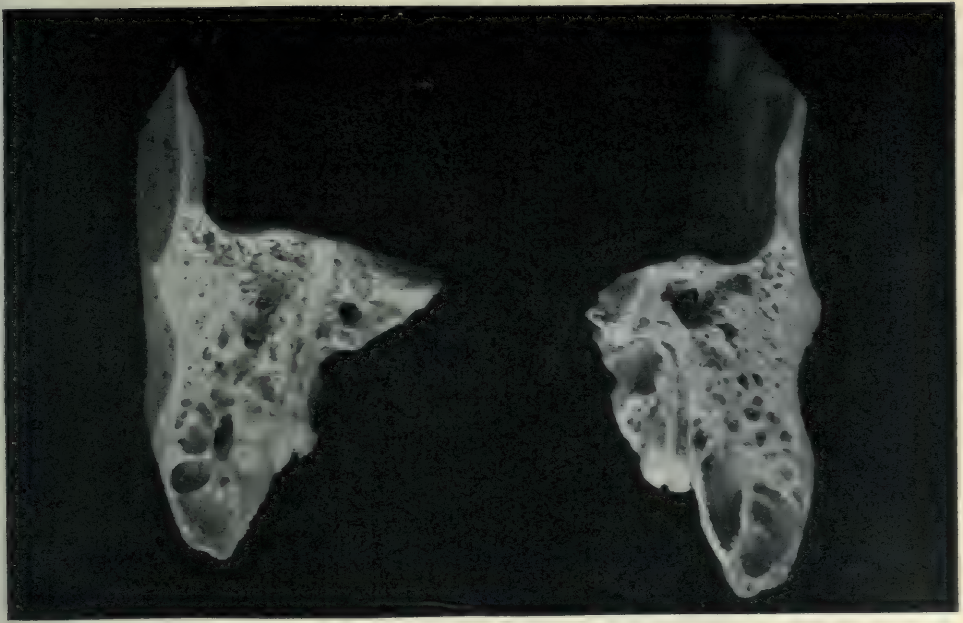
Right.—Outer antral wall and entire mastoid cellular. Long, narrow antrum.



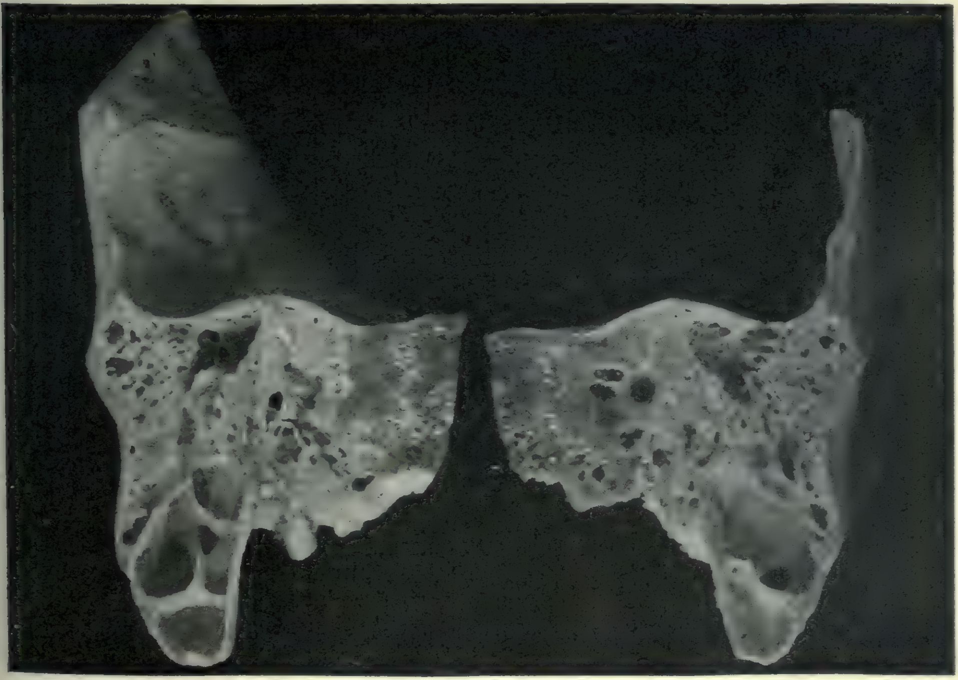
Left.—Symmetrical with the right.

No. 4.—MALE, AGED 50 YEARS.

Symmetrical.



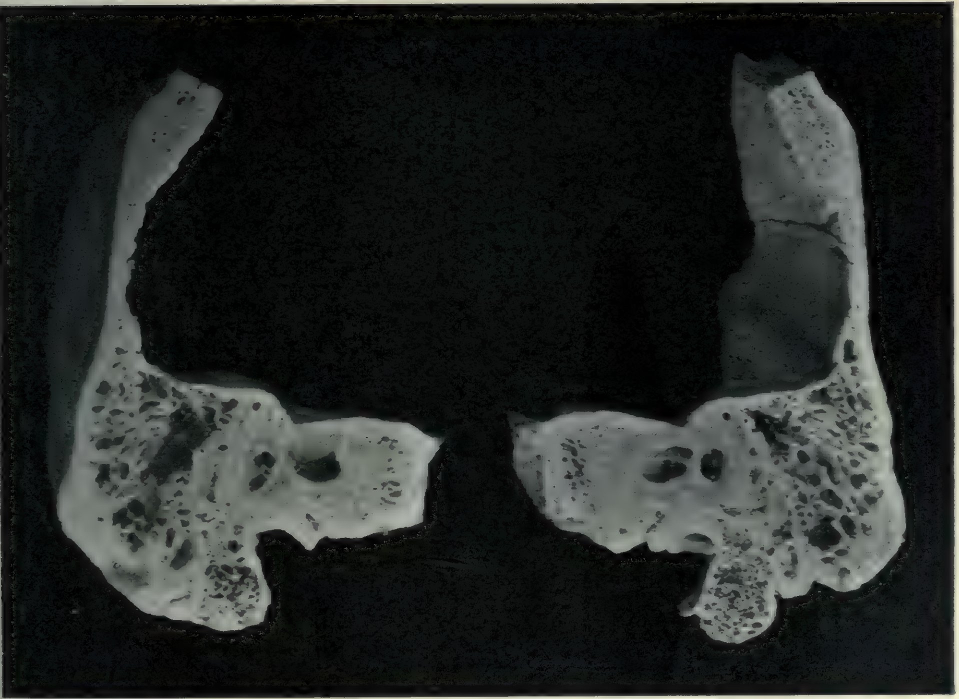
Right.—Outer antral wall and entire mastoid cellular. The lower cells are very large.



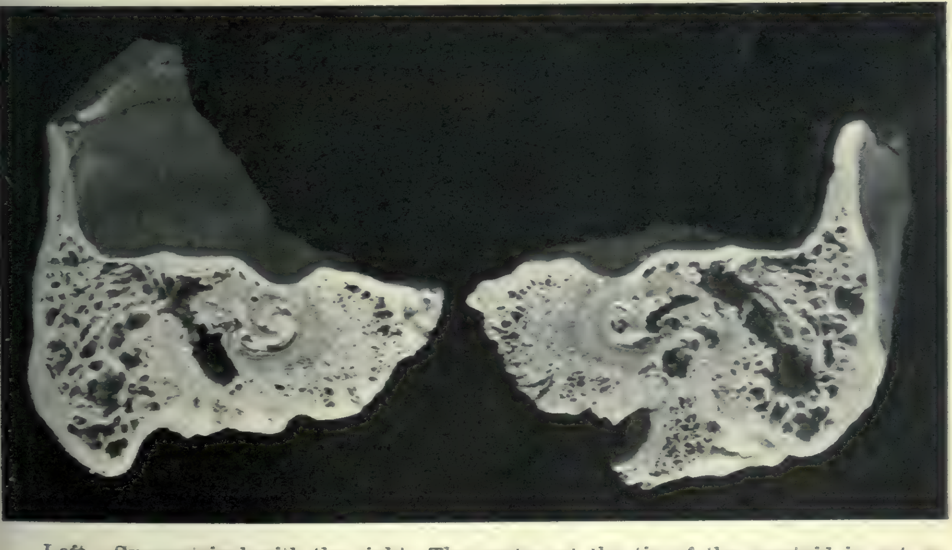
Left.—Symmetrical with the right.

No. 5.—FEMALE, AGED 55 YEARS.

Symmetrical.



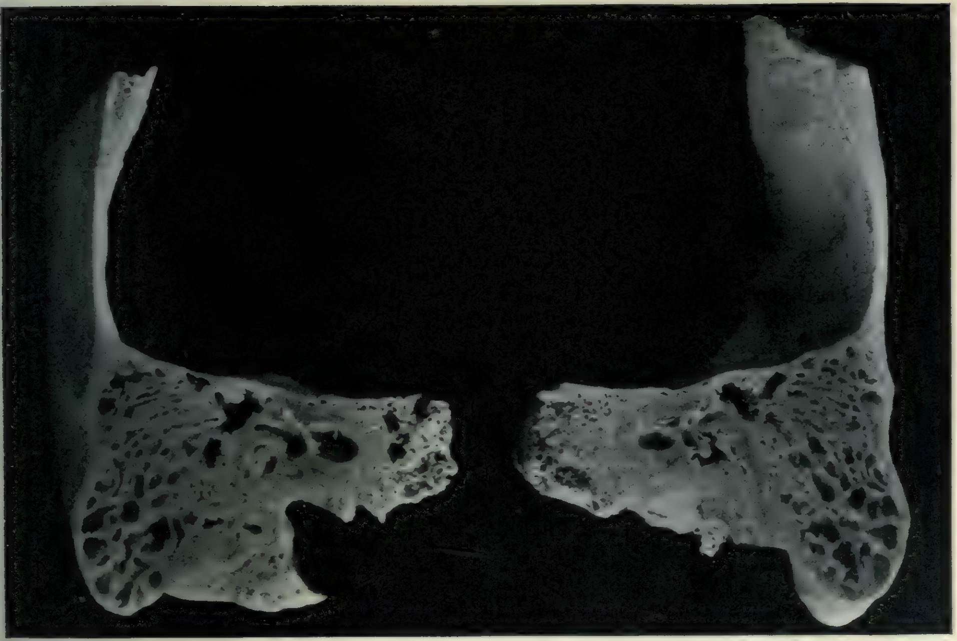
Right.—Outer antral wall and entire mastoid cellular. Long, narrow antrum. The cortex at the tip of the mastoid is very dense.



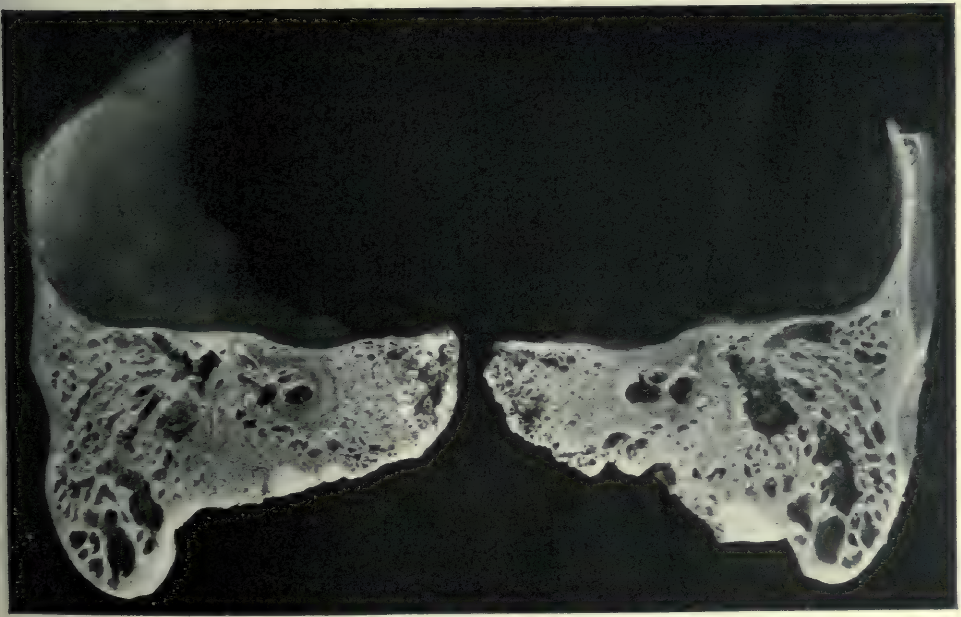
Left.—Symmetrical with the right. The cortex at the tip of the mastoid is not so dense as on the right side.

No. 6.—FEMALE, AGED 62 YEARS.

Symmetrical.



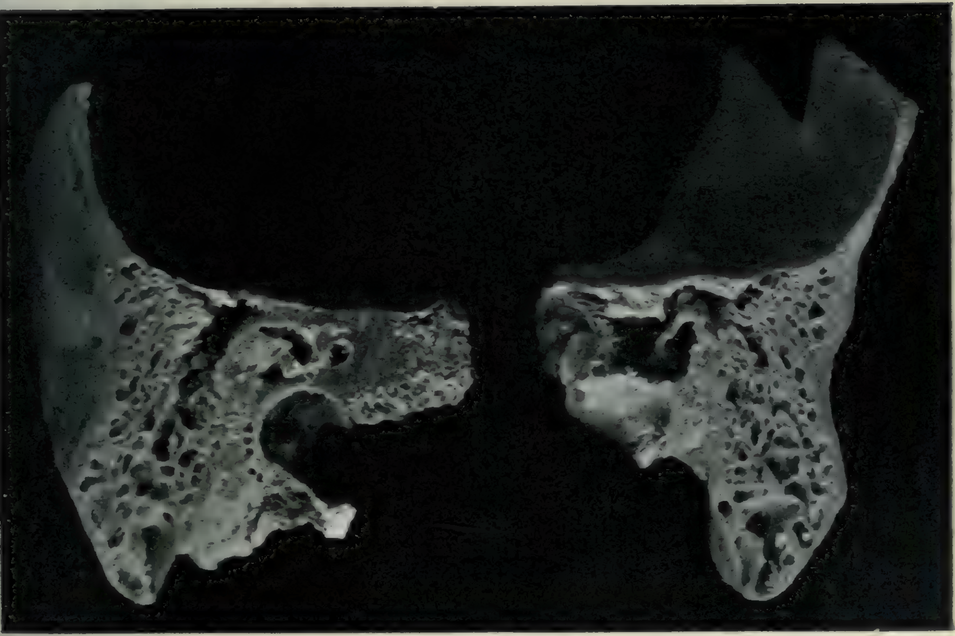
Right.—Outer antral wall and entire mastoid cellular. The antrum is slightly high lying.



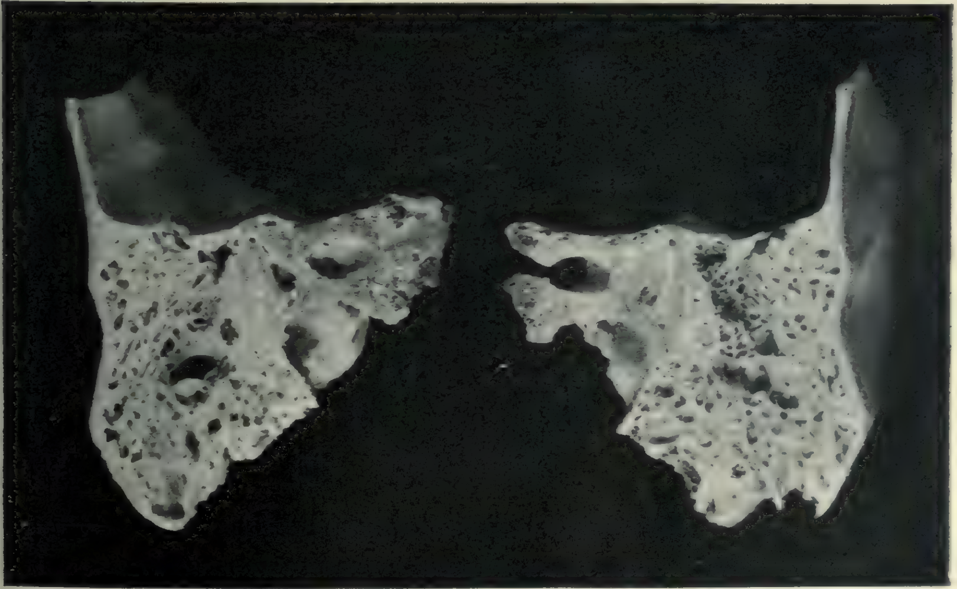
Left.—Symmetrical with the right.

No. 7.—MALE, AGED 41 YEARS.

Symmetrical.



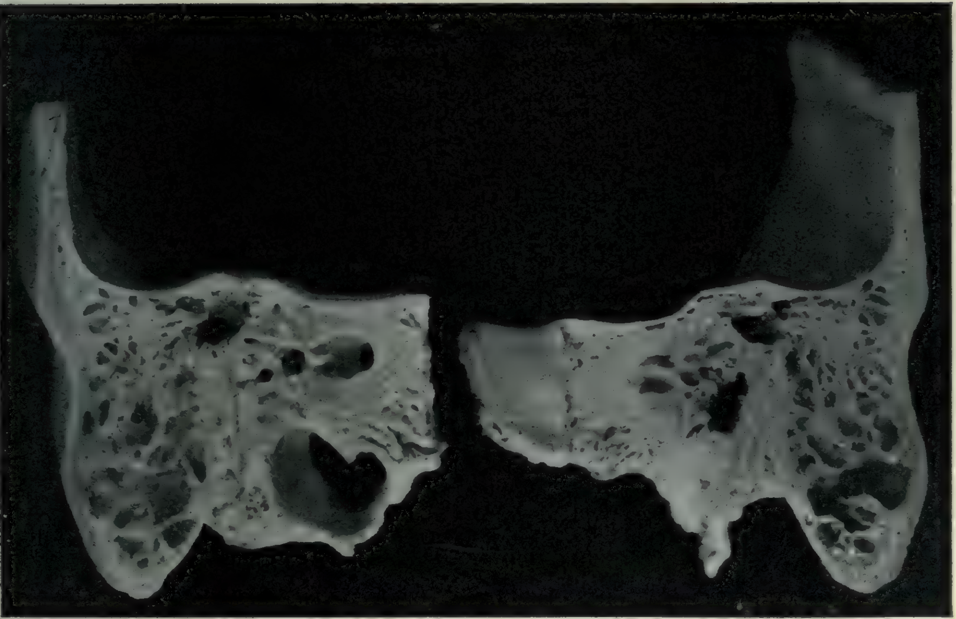
Right.—Outer antral wall and entire mastoid cellular. Long, narrow antrum.



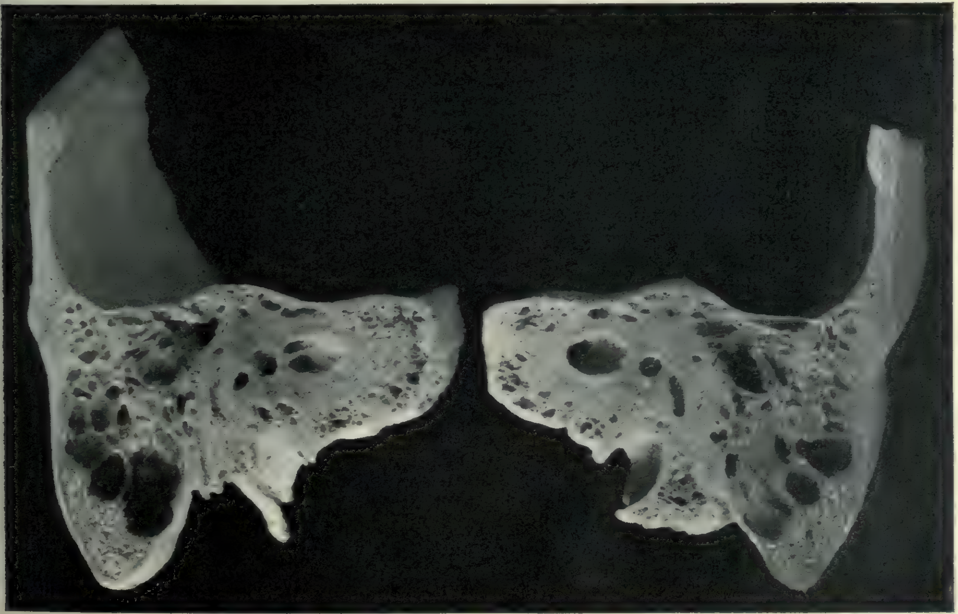
Left.—Symmetrical with the right.

No. 8.—MALE, AGED 36 YEARS.

Slight asymmetry.



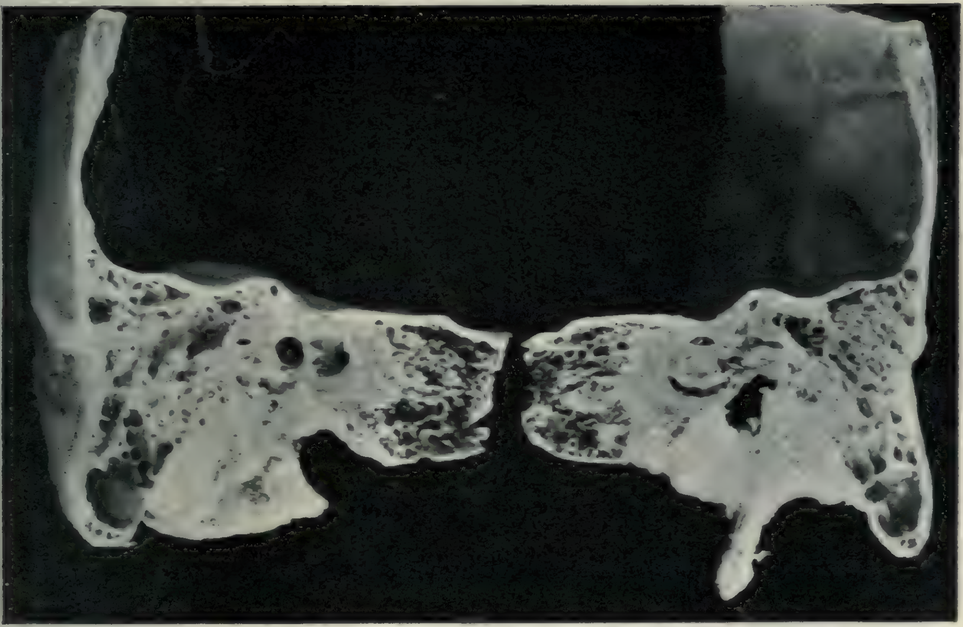
Right.—Outer antral wall and mastoid cellular, with a slight rim of diploe at the tip.



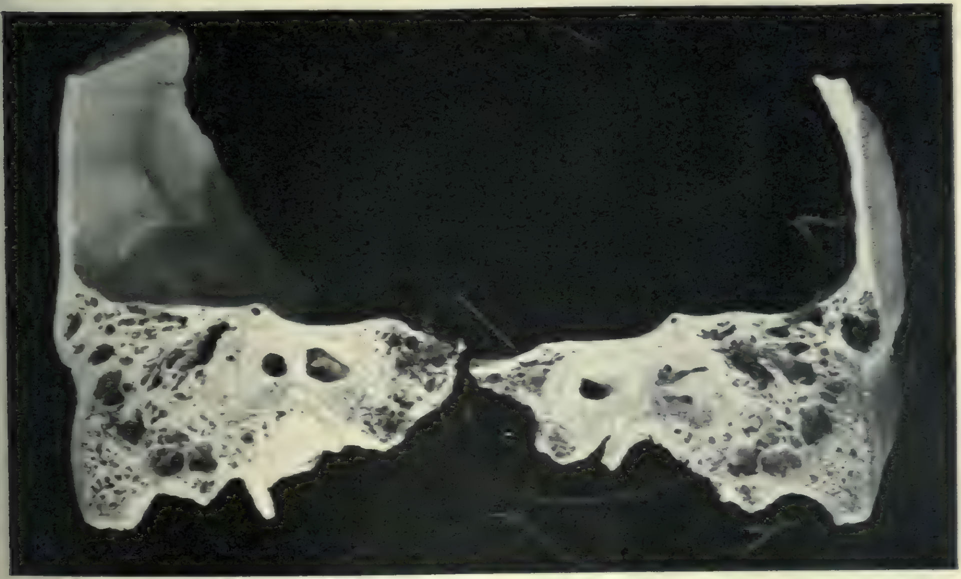
Left.—The same as the right, but there is more diploe at the tip of the mastoid.

No. 9.—MALE, AGED 33 YEARS.

Asymmetrical.

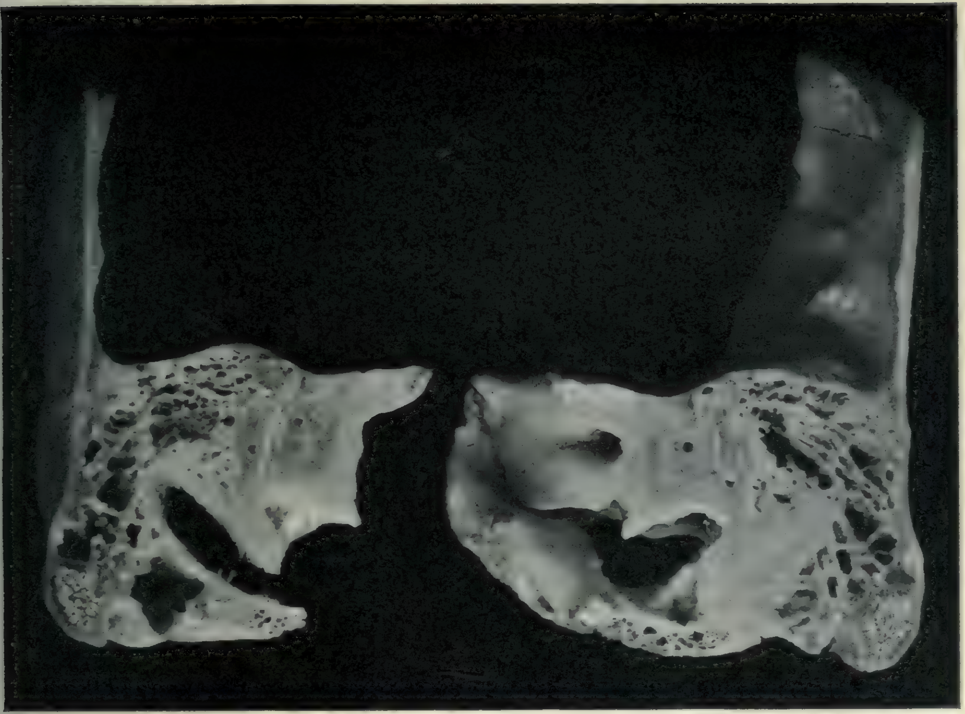


Right.—Outer antral wall and mastoid cellular, with a slight rim of diploe at the tip. Somewhat high-lying antrum.

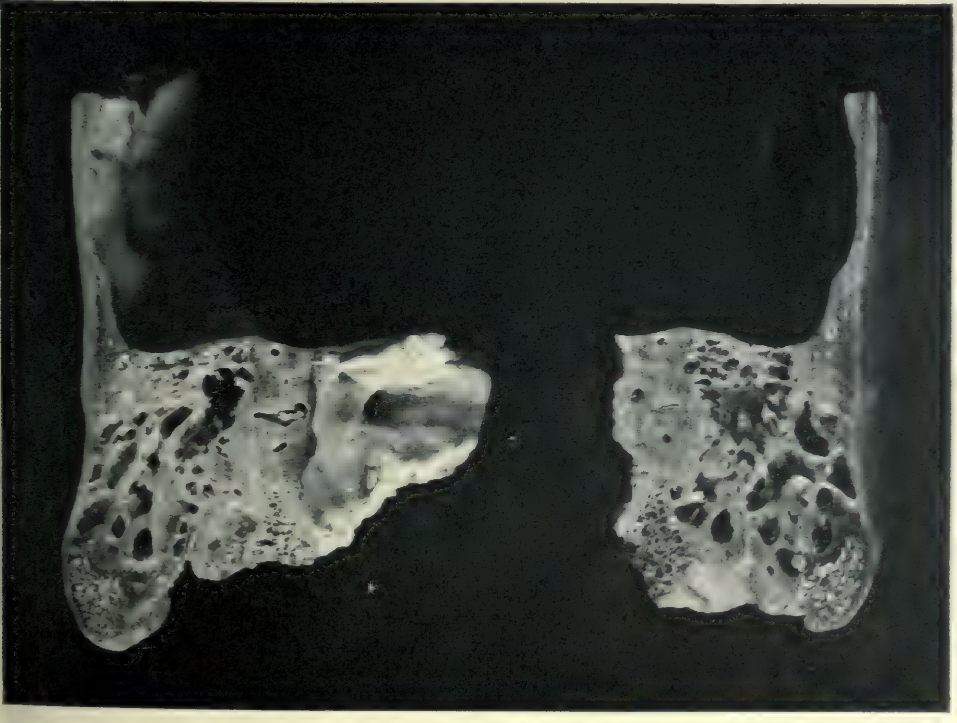


Left.—Outer antral wall and upper mastoid cellular. Lower mastoid is diploetic. There is a specially large cell in the zygomatic element. The cells run downwards and inwards over the digastric fossa. A thick layer of compact bone separates the cells from the diploe. Somewhat high-lying antrum.

Asymmetry of digastric bulla.



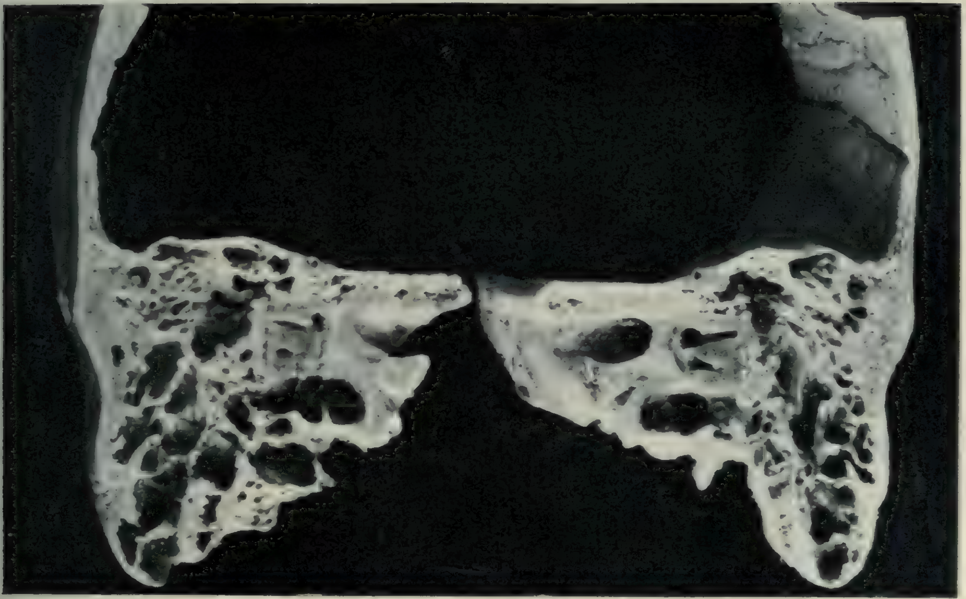
Right.—Outer antral wall partly dense and partly cellular. Upper mastoid cellular. Lower mastoid diploetic. Large digastric bulla.



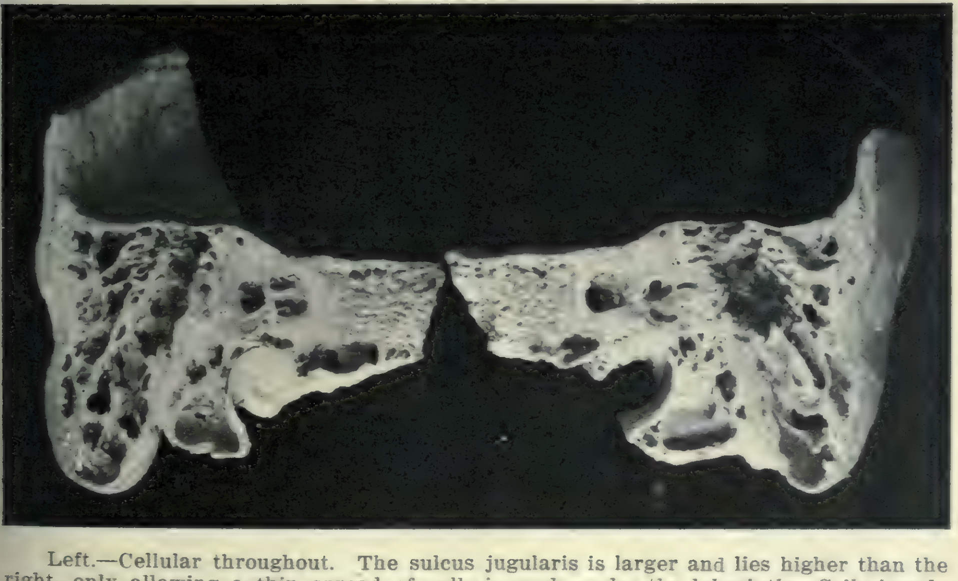
Left.—Symmetrical with the right, except that a smaller digastric bulla is present.

No. 11.—FEMALE, AGED 62 YEARS.

Occipital bulla on the left side only.



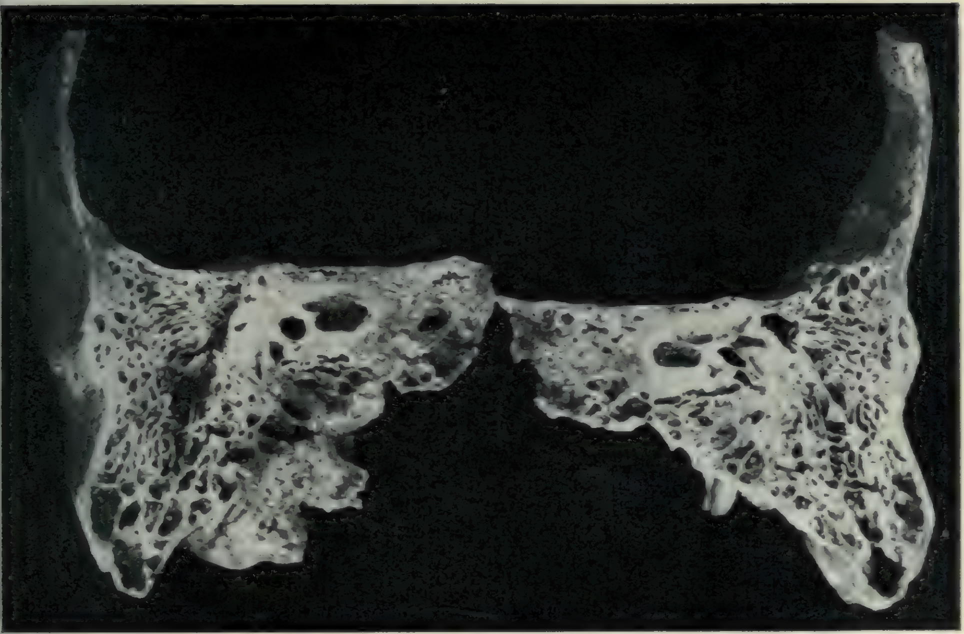
Right.—Cellular throughout. A small, low-lying sulcus jugularis allows the spread of cells inwards under the labyrinth.



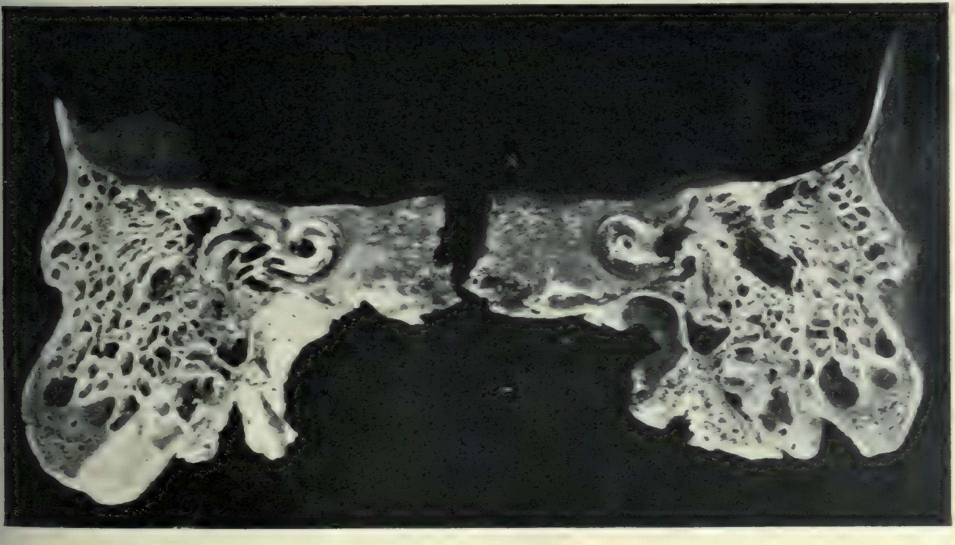
Left.—Cellular throughout. The sulcus jugularis is larger and lies higher than the right, only allowing a thin spread of cells inwards under the labyrinth. Cells invade the occipital, where a larger occipital bulla is present, lying against the outer wall of the sulcus jugularis.

No. 12.—MALE, AGED 51 YEARS.

Left sulcus jugularis the larger.



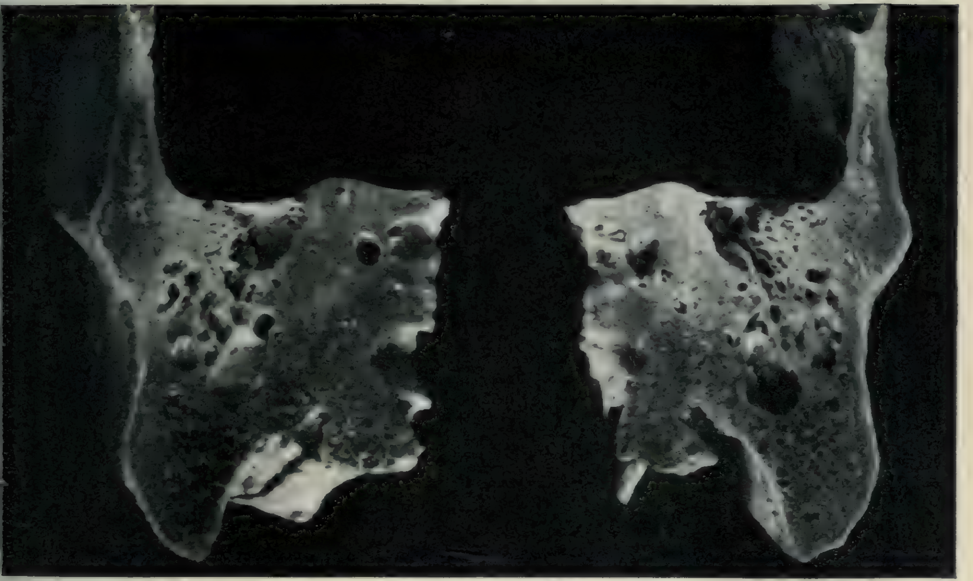
Right.—Cellular throughout. Long, narrow antrum. The cells extend inwards behind the middle ear, over a small, low-lying sulcus jugularis, and under the semicircular, vestibular and internal auditory meatus, and invade the lower part of the internal diploetic mass. The antrum is high lying to the posterior zygomatic line.



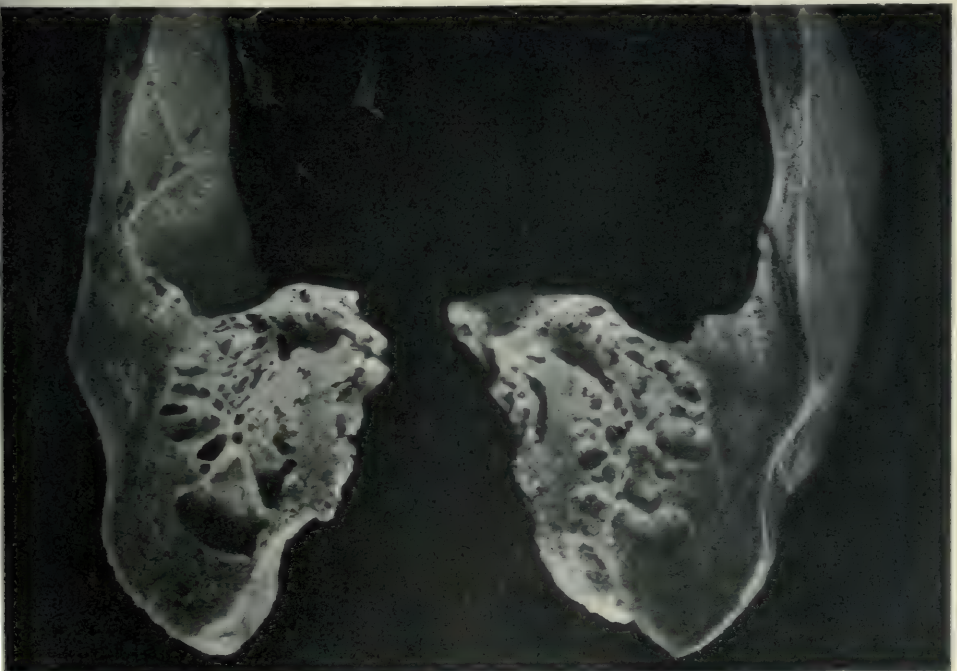
Left.—The antrum is not as long as on the right side, and diploe is present in the tip of the mastoid. The sulcus jugularis is larger and lies higher then on the right, lessening the cellular spread inwards. The cells run up to its outer wall. A digastric bulla is present. The antrum is high-lying.

No. 13.—MALE, AGED 50 YEARS.

Dipping down of the middle fossa on one side only, the left.



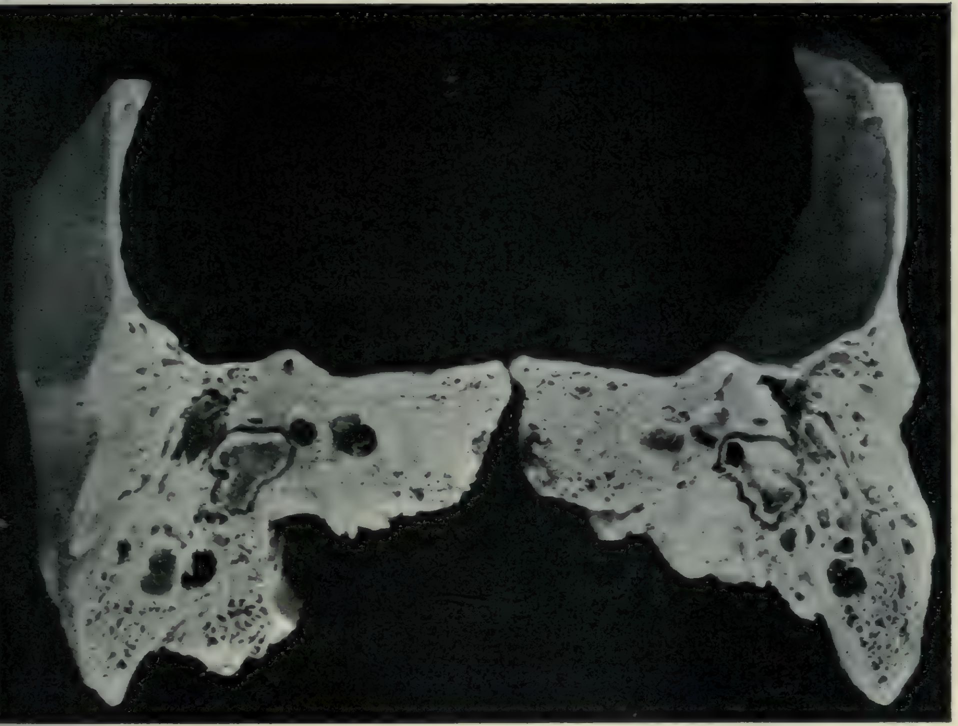
Right.—Outer antral wall dense. Upper mastoid cellular. Lower mastoid diploetic.



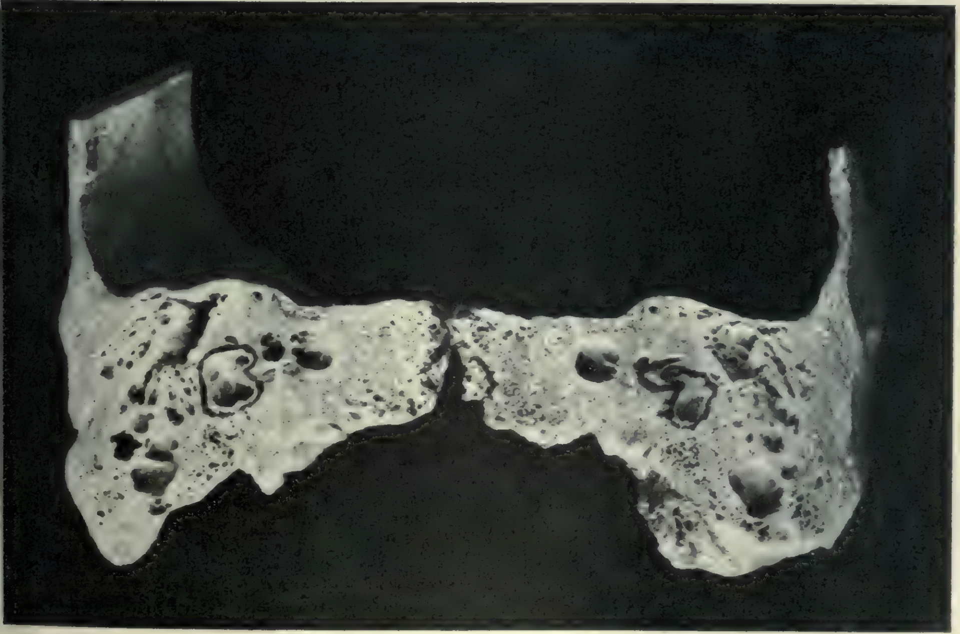
Left.—Interior symmetrical with the right, but the middle fossa dips down, causing a sloping roof to the antrum, a condition which is not present on the right side.

No. 14.—MALE, AGED 58 YEARS.

Symmetrical.



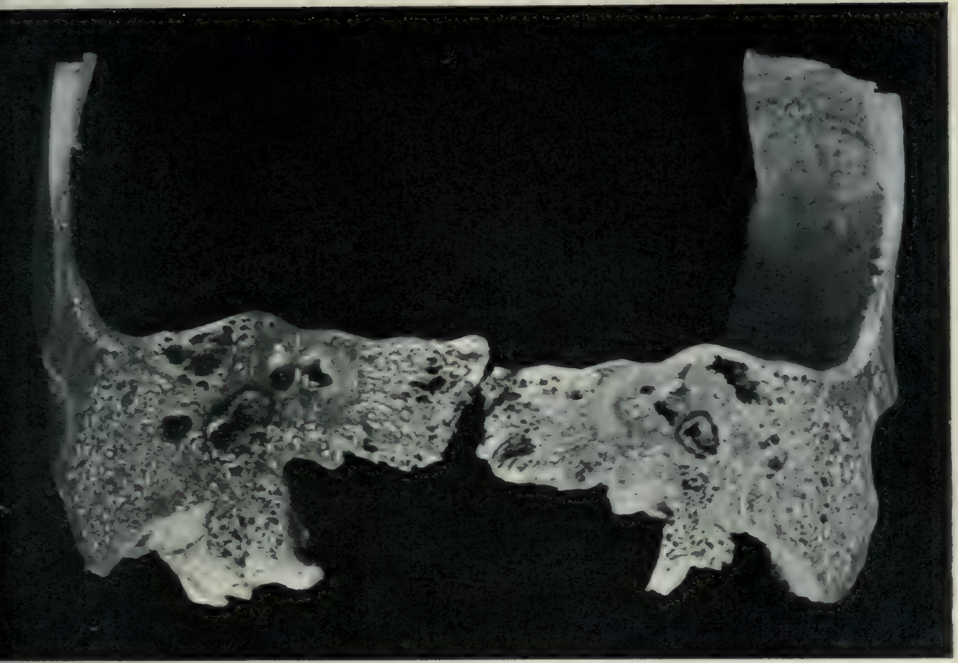
Right.—Showing an extensive sinus tympani (outlined in ink) with a small opening into the posterior middle ear wall.



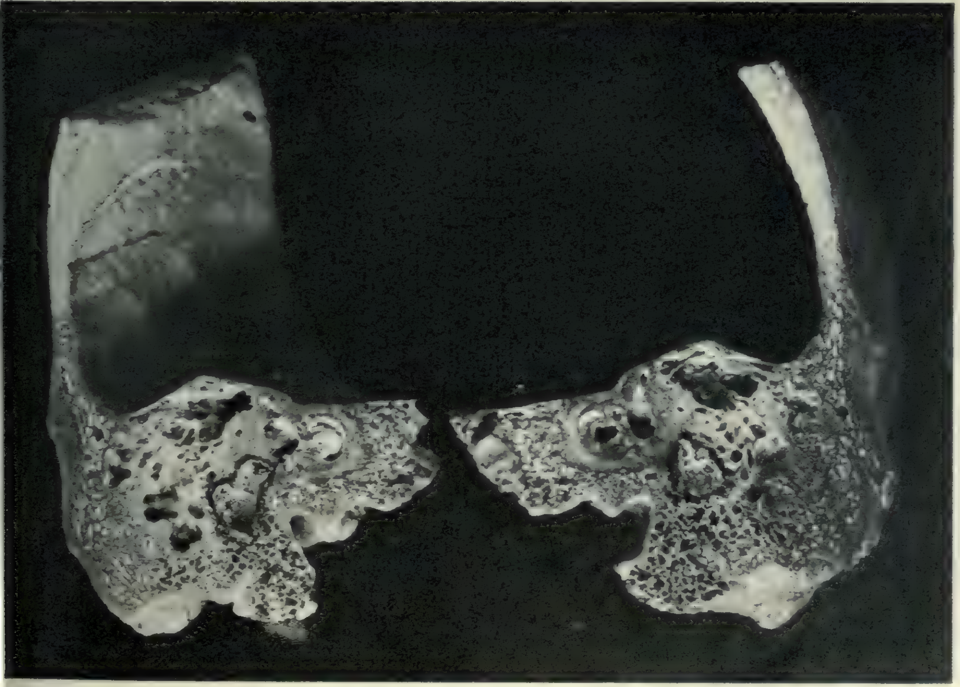
Left.—Symmetrical with the right.

No. 15.—MALE, AGED 64 YEARS.

Symmetrical.

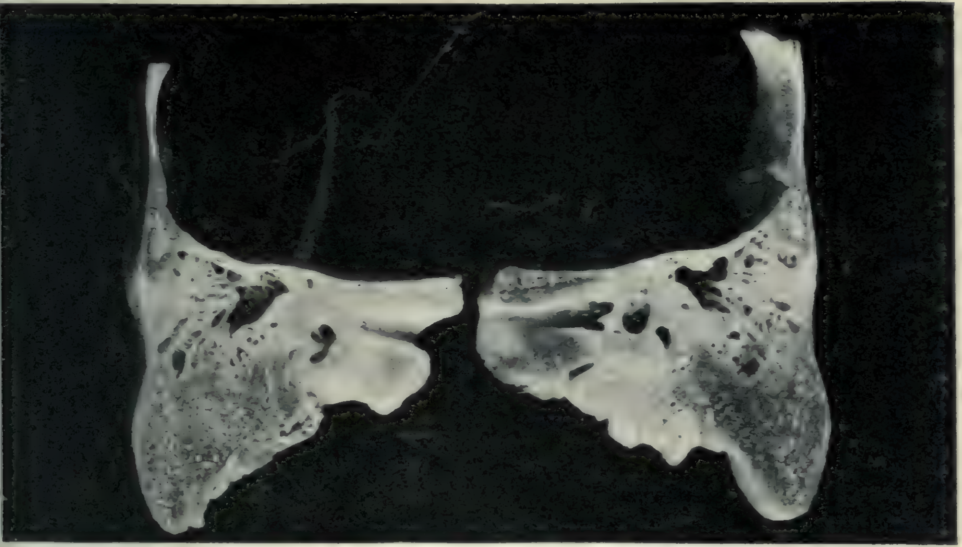


Right.—Showing a large sinus tympani, outlined in ink.

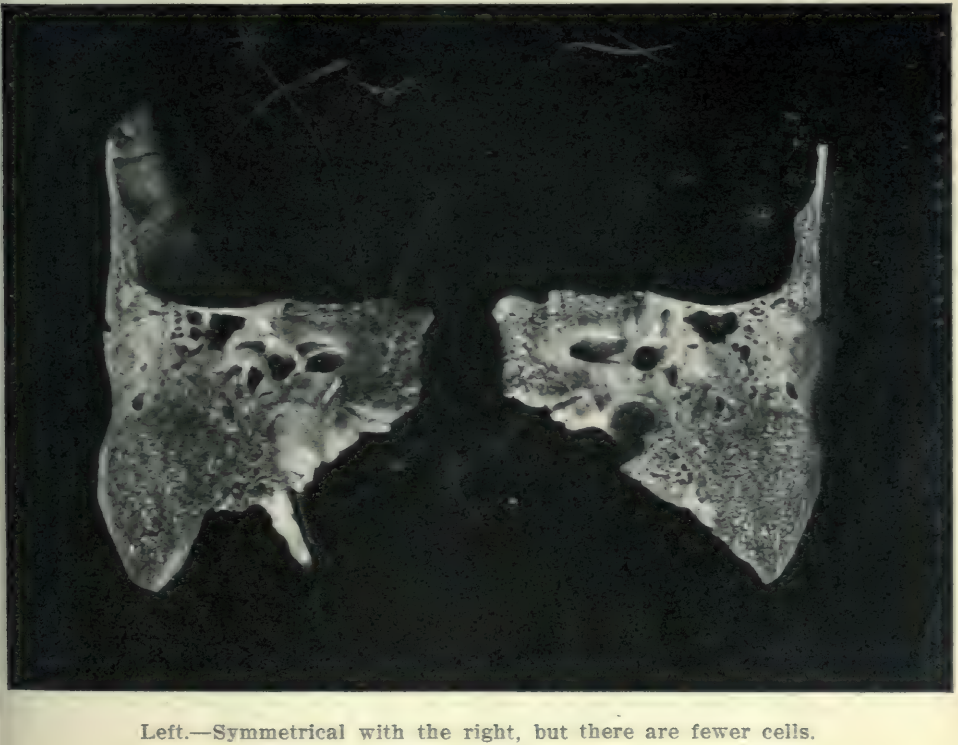


Left.—Symmetrical sinus tympani.

No. 16.—FEMALE, AGED 55 YEARS.



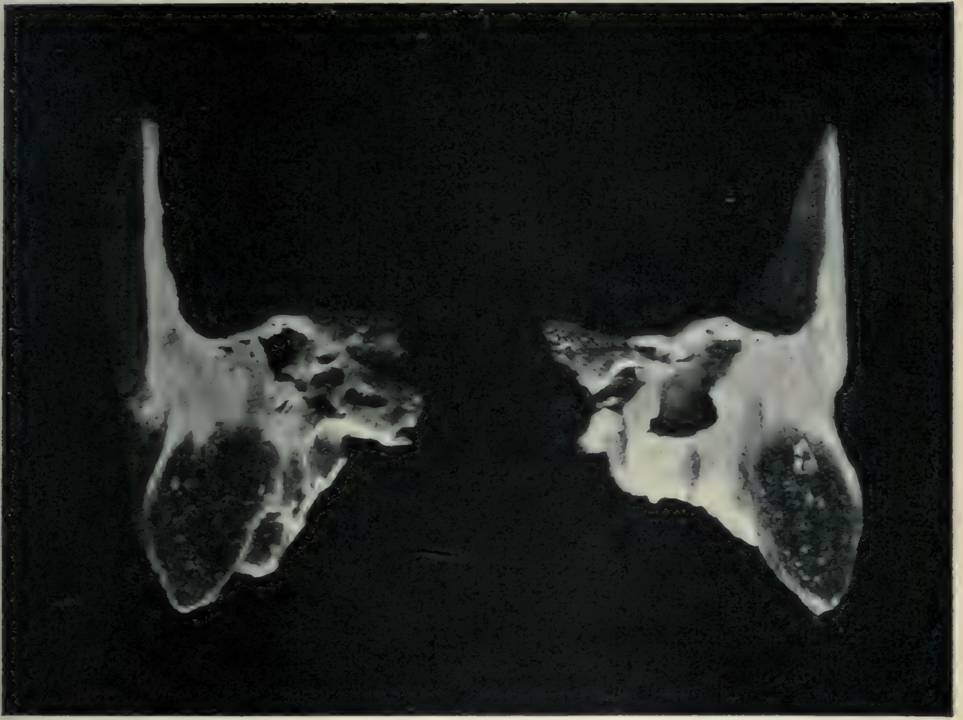
Right.—A few cells are present in the dense outer antral wall and in the upper part of the diploetic mastoid.



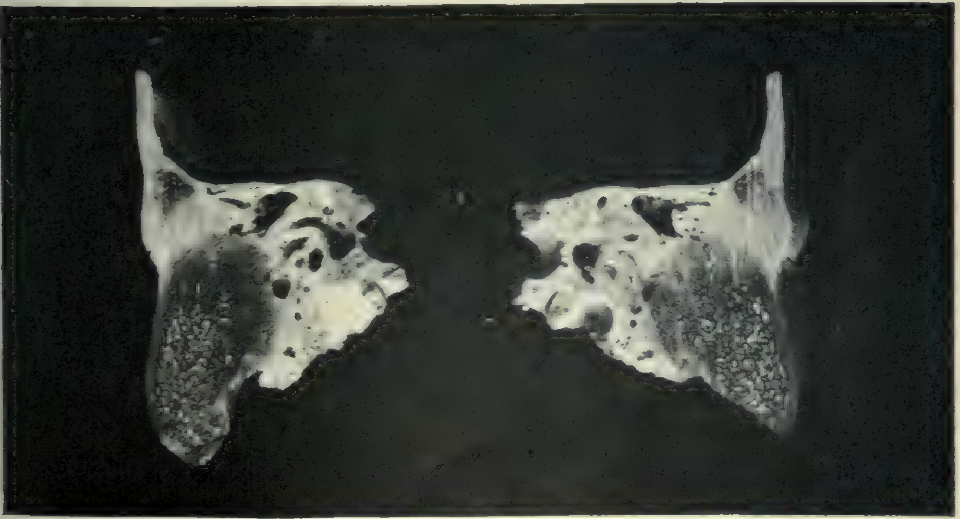
Left.—Symmetrical with the right, but there are fewer cells.

No. 17.—MALE, AGED 28 YEARS.

Symmetrical diploetic infantile type.



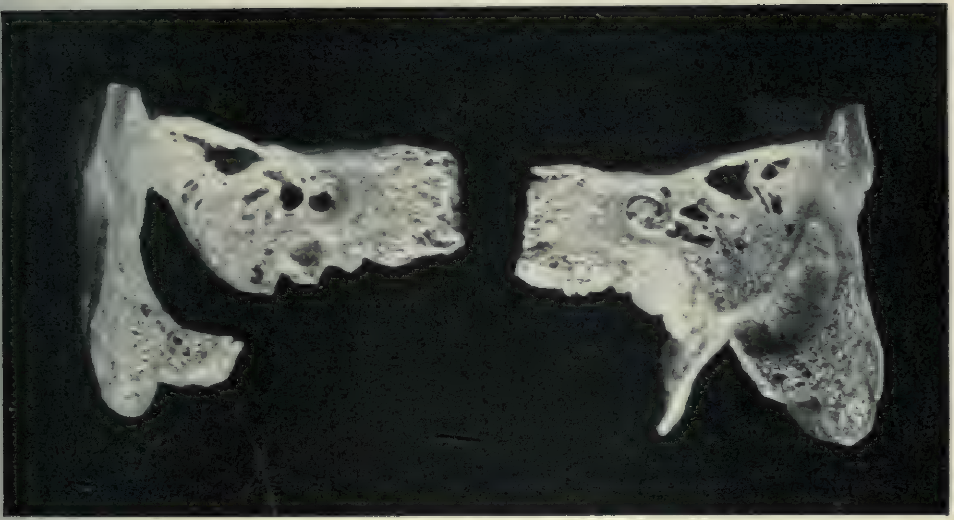
Right.—Diploetic infantile type.



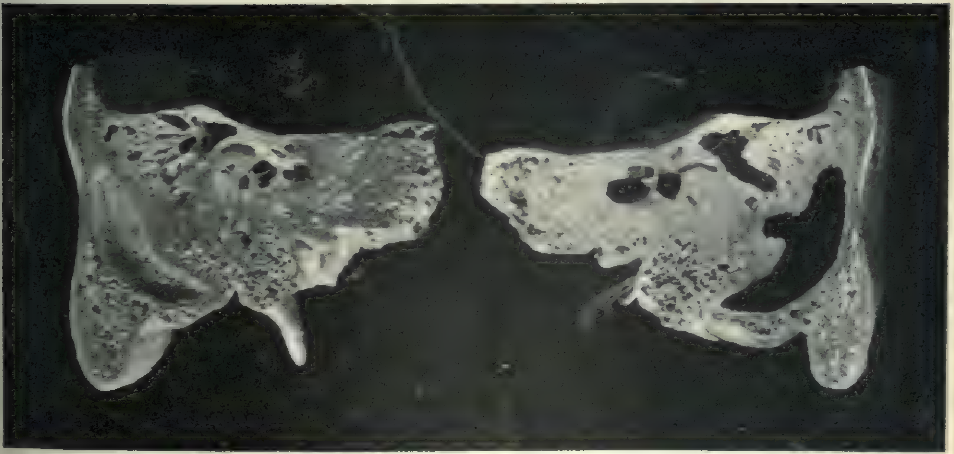
Left.—Diploetic infantile type, symmetrical with right.

No. 18.—MALE, AGED 45 YEARS.

Symmetrical diploetic infantile type.



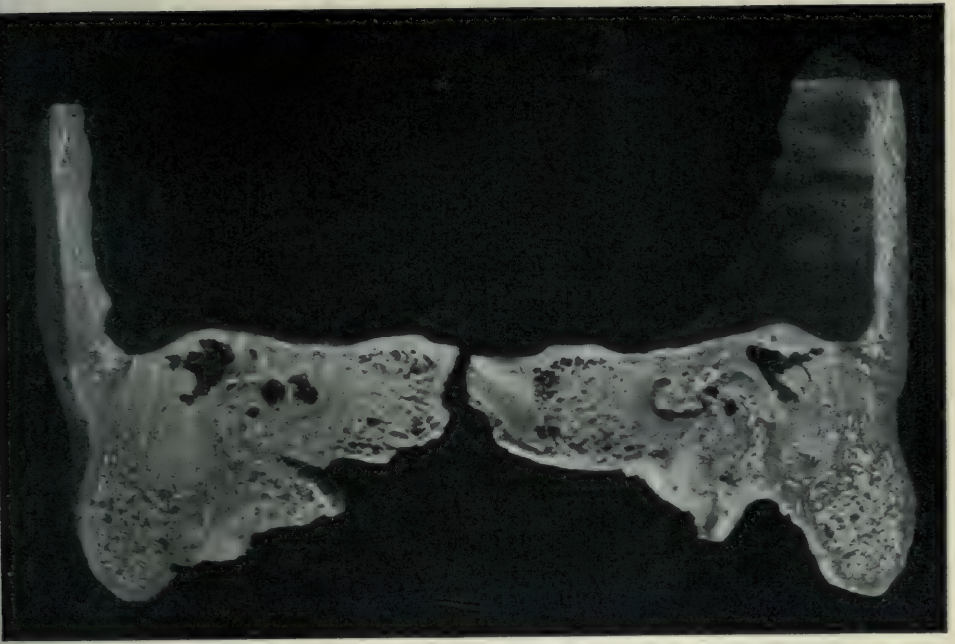
Right.—With very forward lateral sinus.



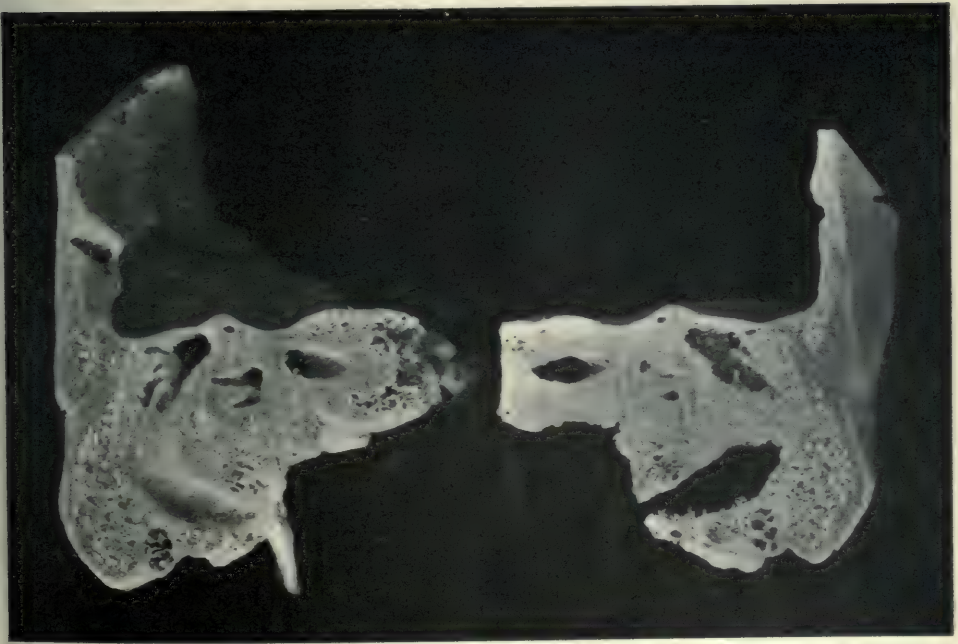
Left.—With very forward lateral sinus.

No. 19.—MALE, AGED 58 YEARS.

Symmetrical diploetic infantile type.



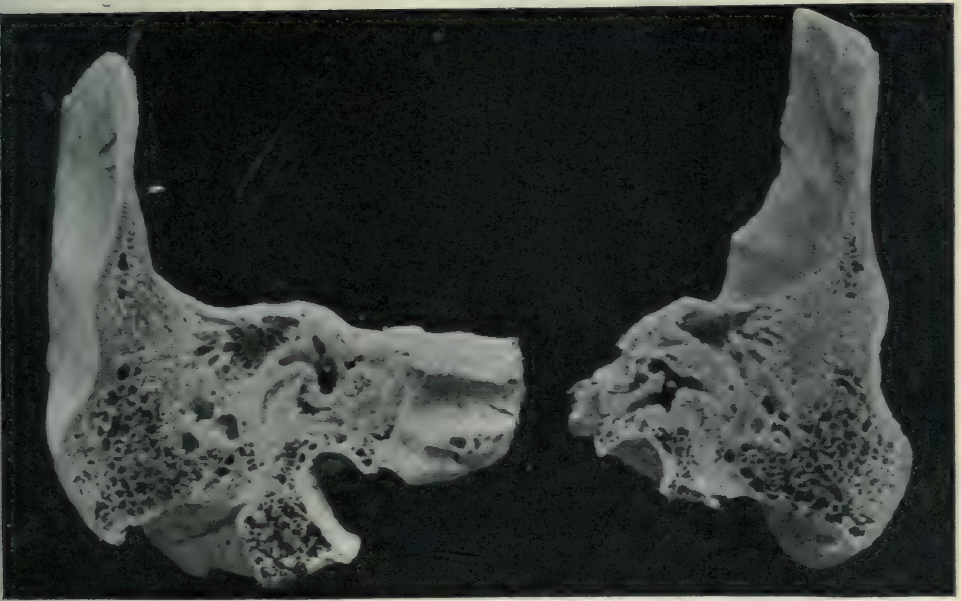
Right.—Bilateral diploetic type with dense outer antral wall and a dense layer between the antrum and diploe of the mastoid.



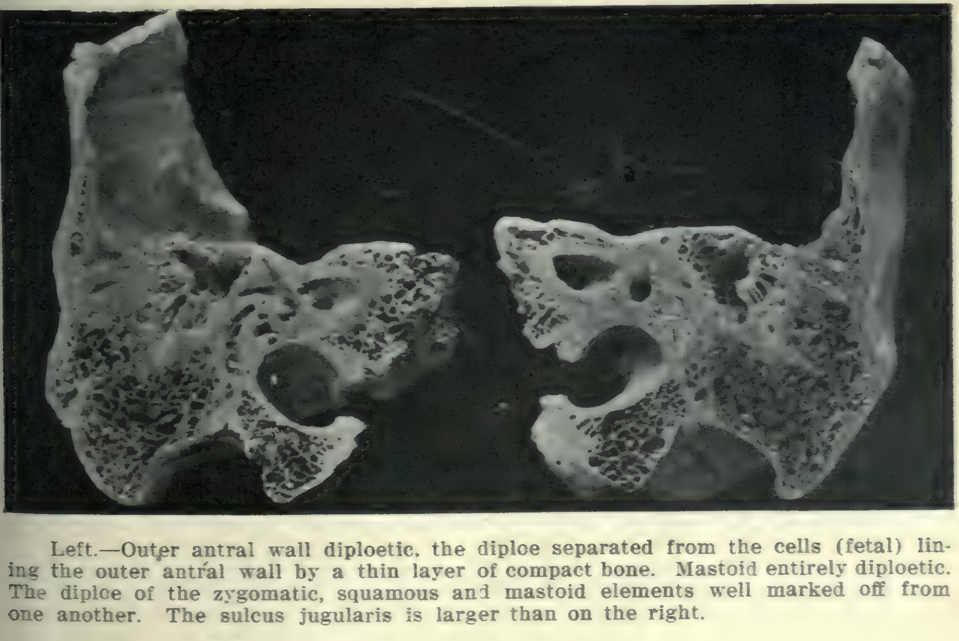
Left.—Symmetrical with right side.

No. 20.—MALE, AGED 33 YEARS.

Congenital imbecile.

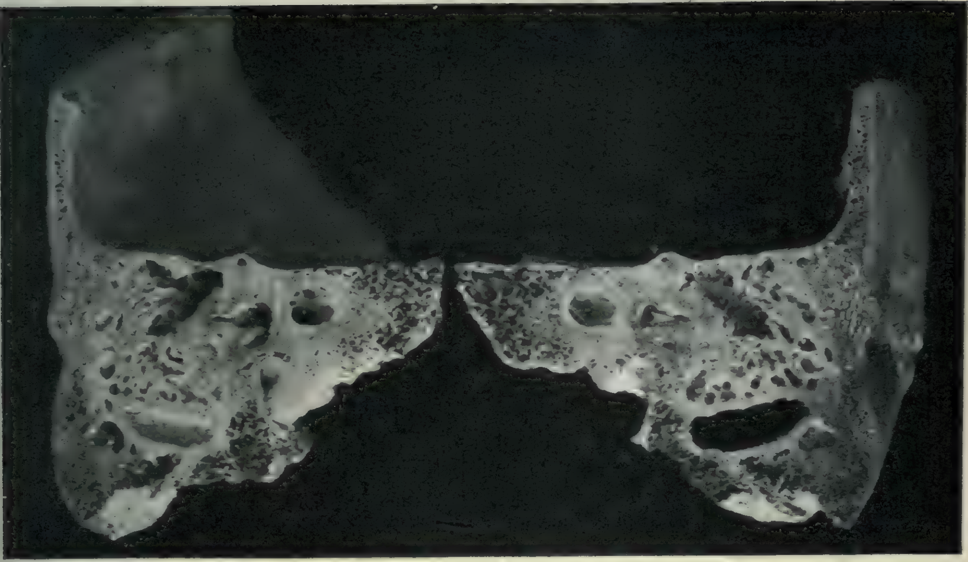


Right.—Outer antral wall dense. A few cells at the upper and inner aspect of a diploetic mastoid.

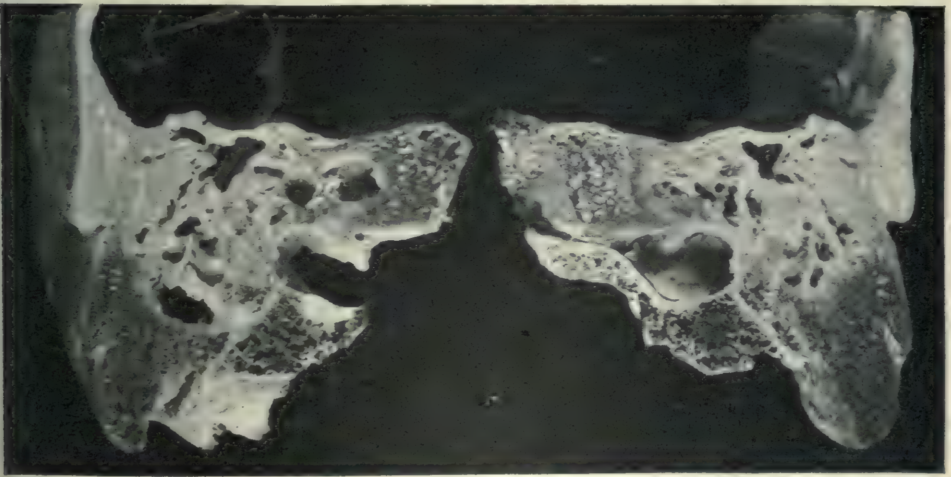


Left.—Outer antral wall diploetic, the diploe separated from the cells (fetal) lining the outer antral wall by a thin layer of compact bone. Mastoid entirely diploetic. The diploe of the zygomatic, squamous and mastoid elements well marked off from one another. The sulcus jugularis is larger than on the right.

No. 21.—MALE, AGED 37 YEARS.

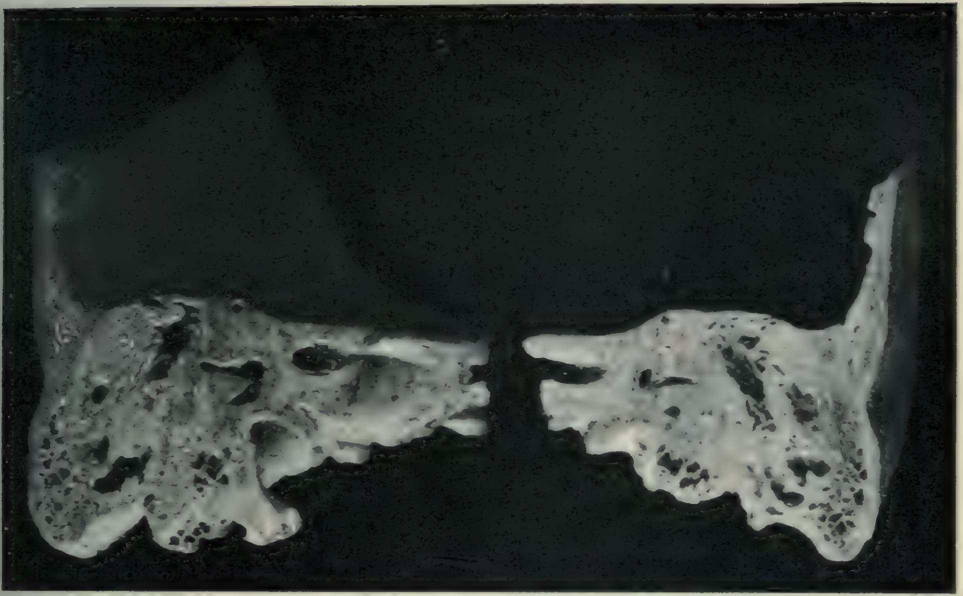


Right.—Outer antral wall dense. A few cells in the upper mastoid. Diploetic lower mastoid. High-lying antrum with an overhanging posterior zygomatic line.

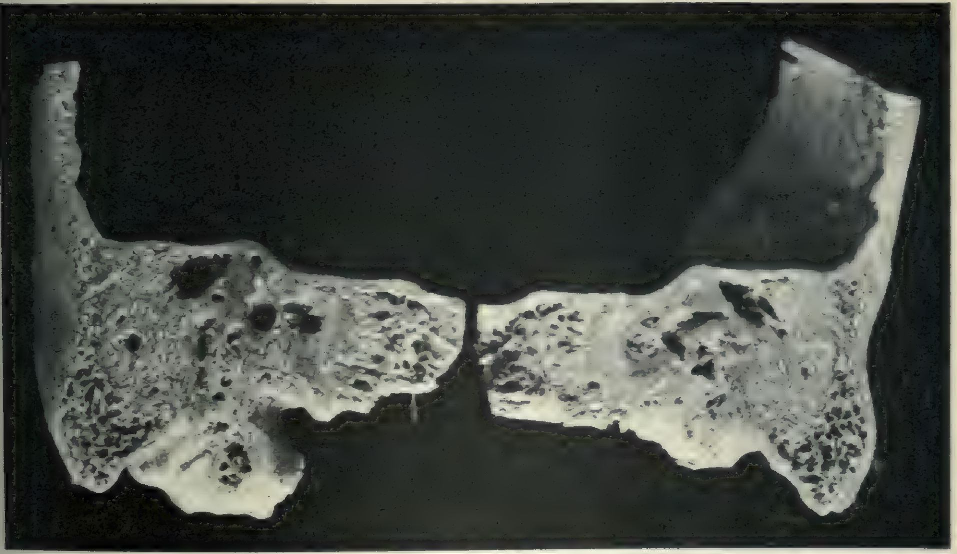


Left.—Outer antral wall has a thin layer of diploe running through it. Cellular upper mastoid. Diploetic lower mastoid. The anterior section shows the marking off of the zygomatic, squamous and petrous elements. High-lying antrum.

No. 22.—FEMALE, AGED 33 YEARS.



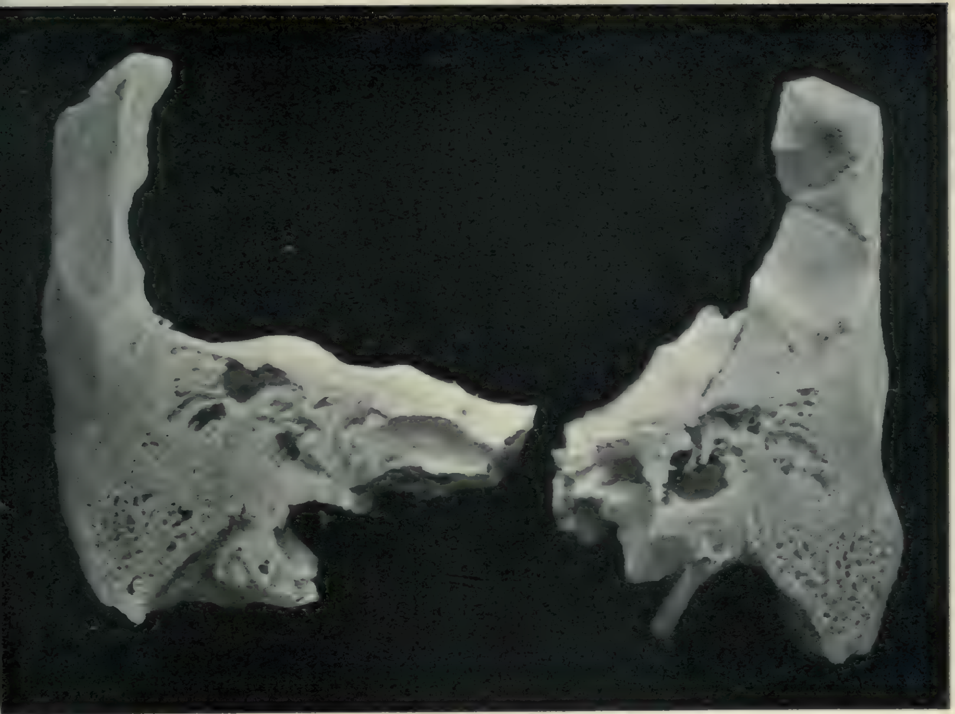
Right.—Outer antral wall dense. A few cells in the inner aspect of the upper mastoid. Diploetic lower mastoid.



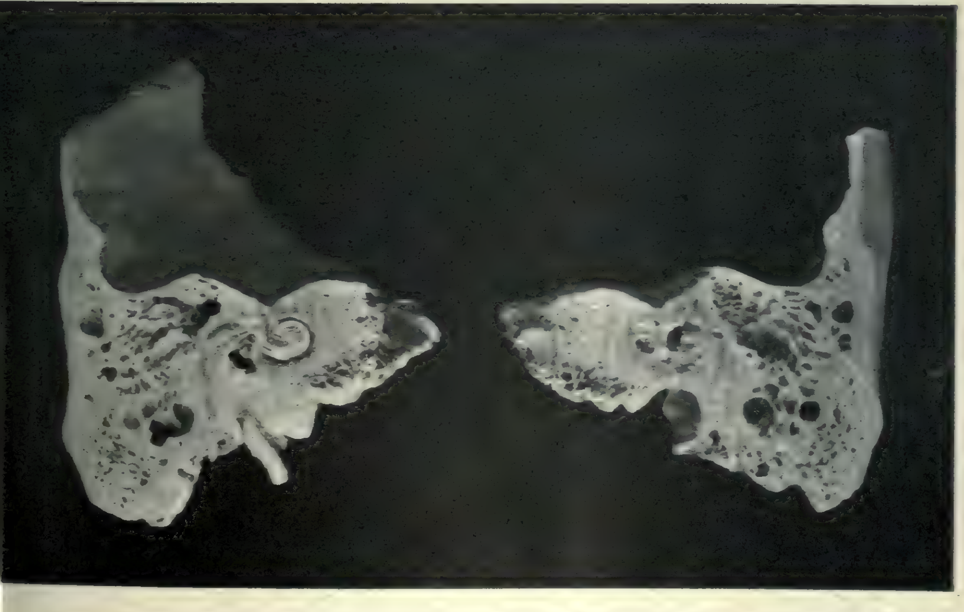
Left.—A thin layer of diploe in the outer antral wall continuous with the diploe of the mastoid. A few cells in the upper part of the upper mastoid.

No. 23.—MALE, AGED 39 YEARS.

Gross asymmetry.



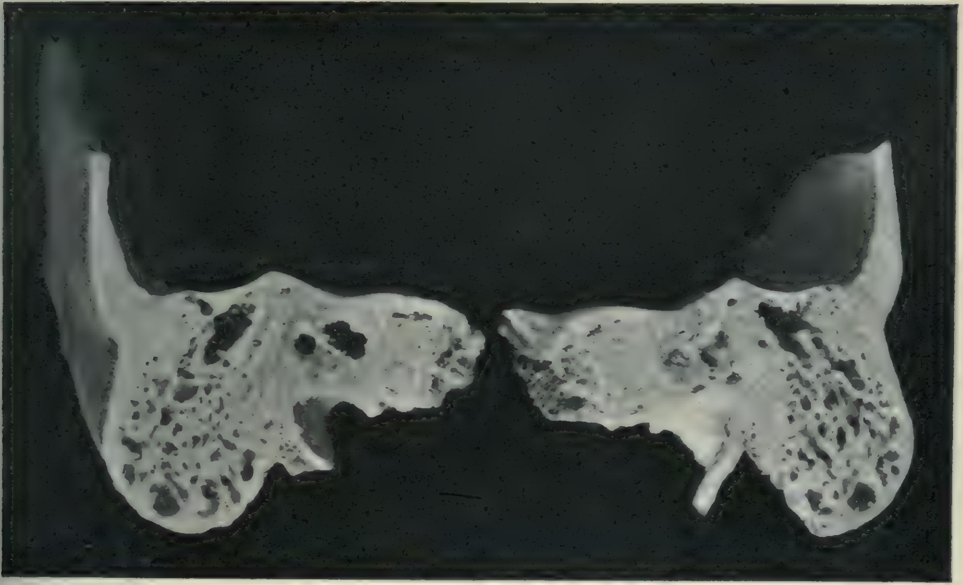
Right.—Diploetic infantile type.



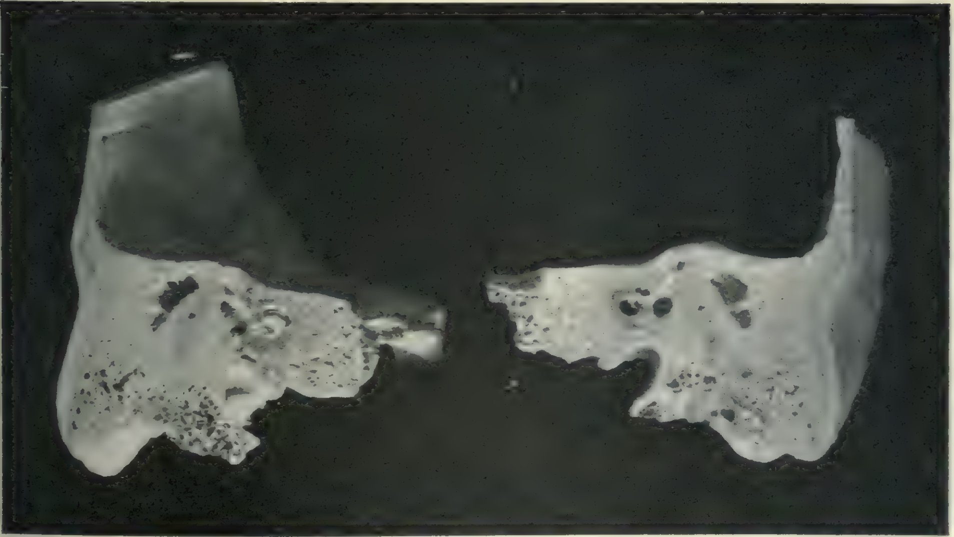
Left.—Outer antral wall and upper mastoid cellular. Lower mastoid diploetic. Dipping down of the middle fossa, causing a sloping roof to the antrum, a condition not seen on the right side.

No. 24.—FEMALE, AGED 41 YEARS.

Gross asymmetry.



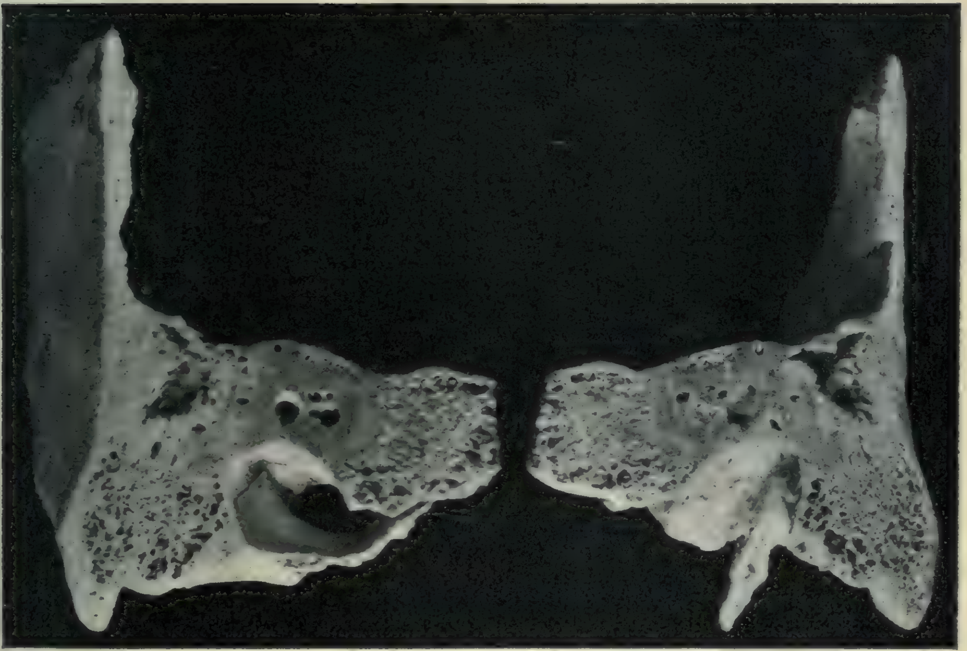
Right.—Outer antral wall dense. Mastoid finely cellular throughout.



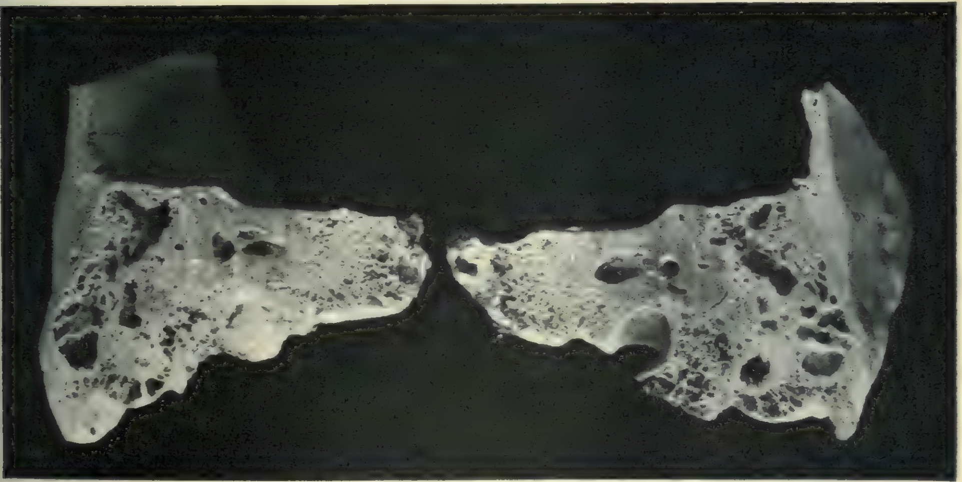
Left.—Diploetic infantile type.

No. 25.—FEMALE, AGED 48 YEARS.

Gross asymmetry.



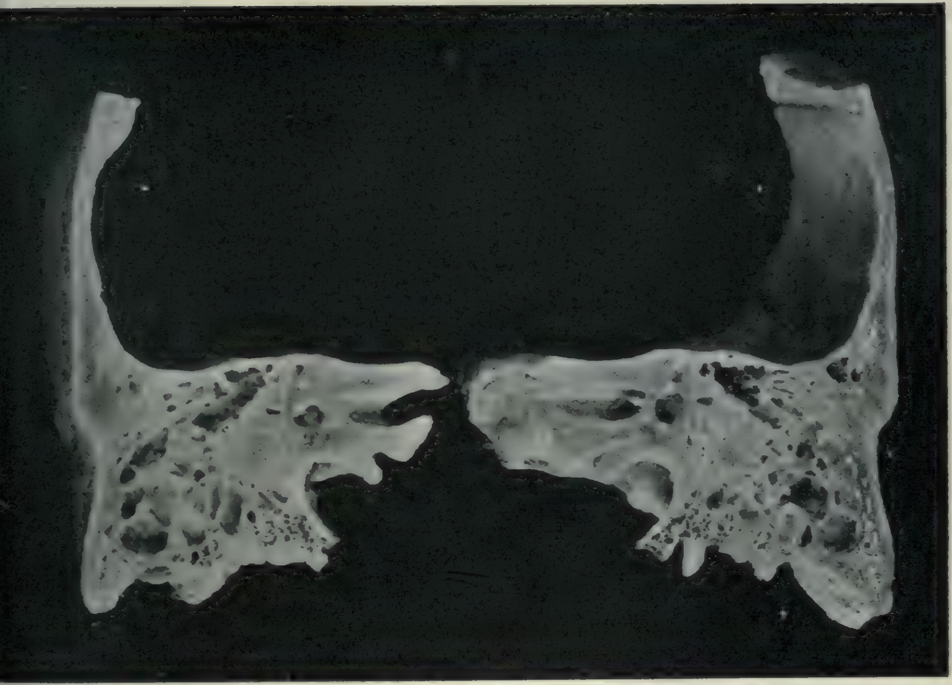
Right.—Diploetic infantile type.



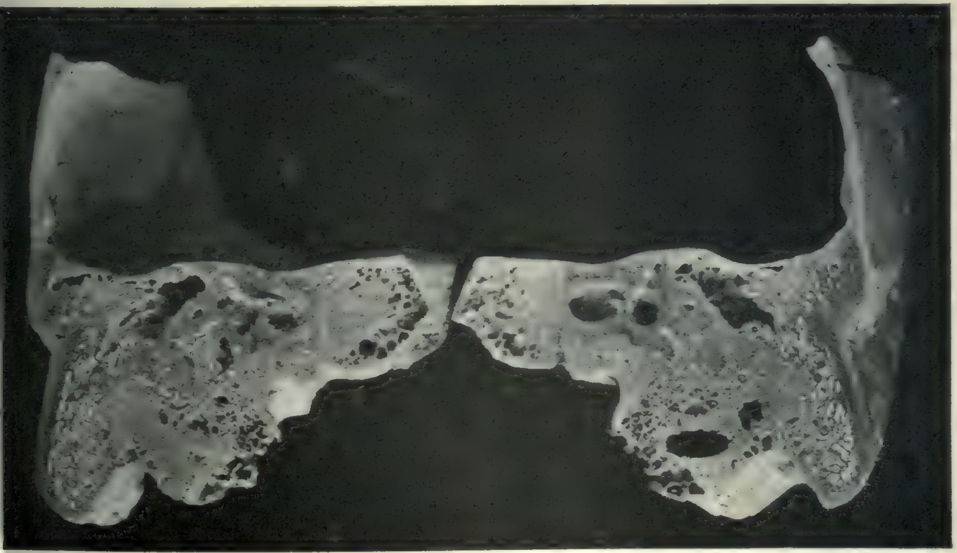
Left.—Dense outer antral wall. Cellular upper mastoid. Diploetic lower mastoid.

No. 26.—MALE, AGED 49 YEARS.

Gross asymmetry



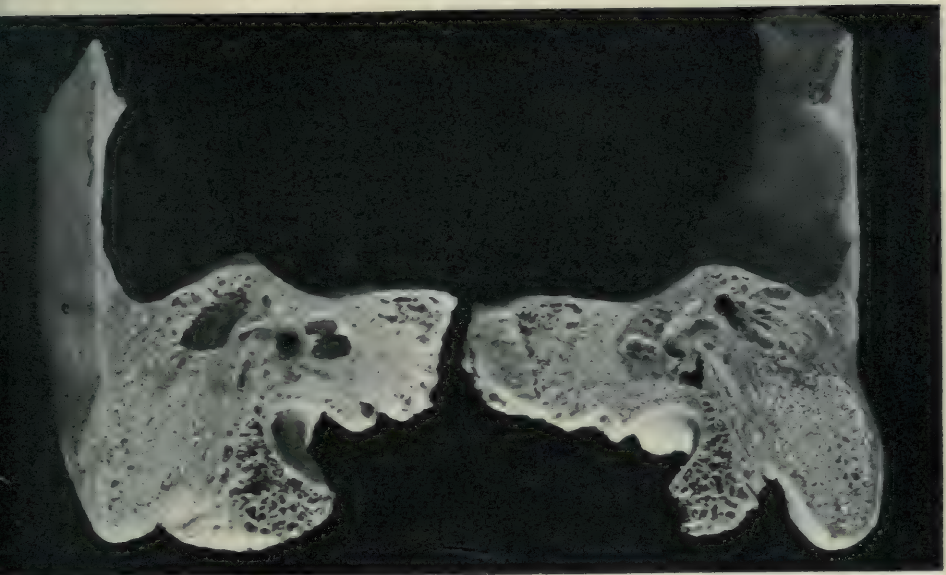
Right.—Outer antral wall dense. Cell in zygomatic diploe. Upper mastoid cel-
lular. Lower mastoid diploetic.



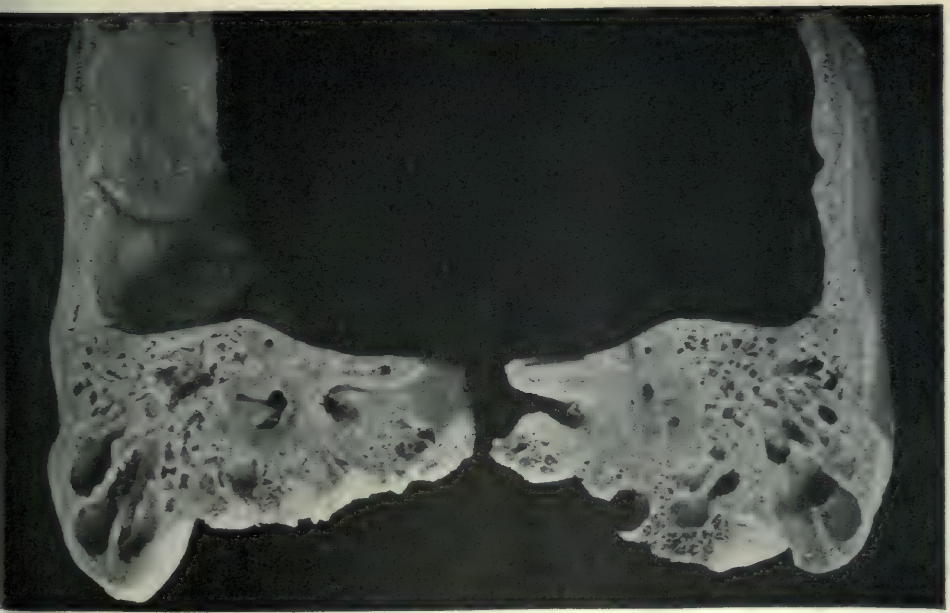
Left.—Diploetic infantile type.

No. 27.—FEMALE, AGED 50 YEARS.

Gross asymmetry.



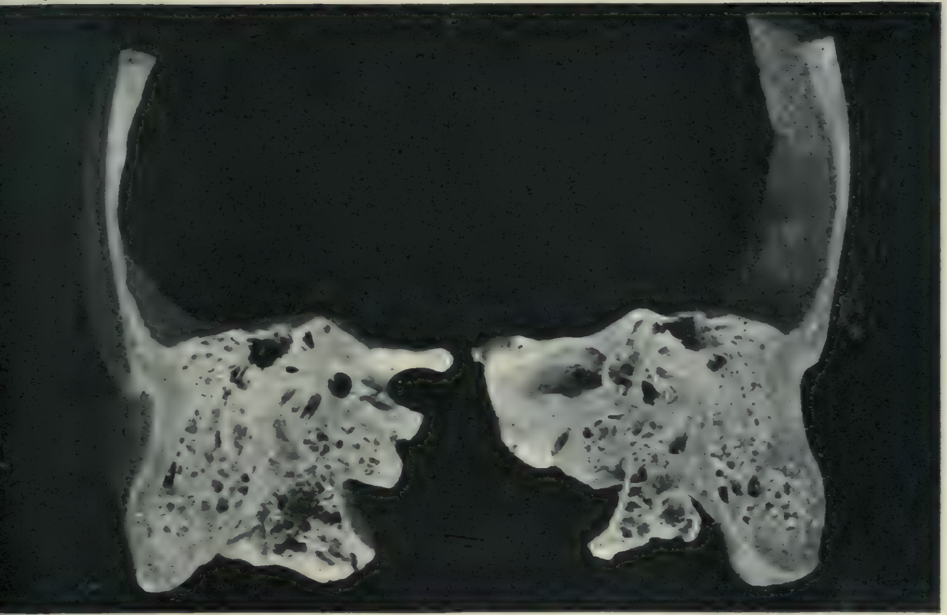
Right.—Diploetic infantile type. Middle fossa dips down, causing a sloping roof o the antrum, a condition not seen on the left side.



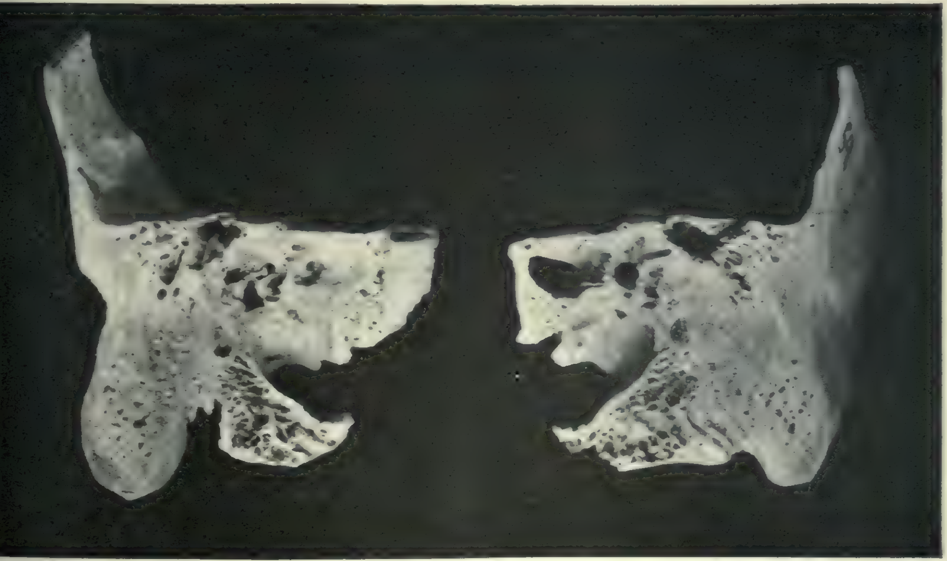
Left.—Cellular outer antral wall and mastoid with a slight rim of diploe at the tip.

No. 28.—MALE, AGED 59 YEARS.

Asymmetry.



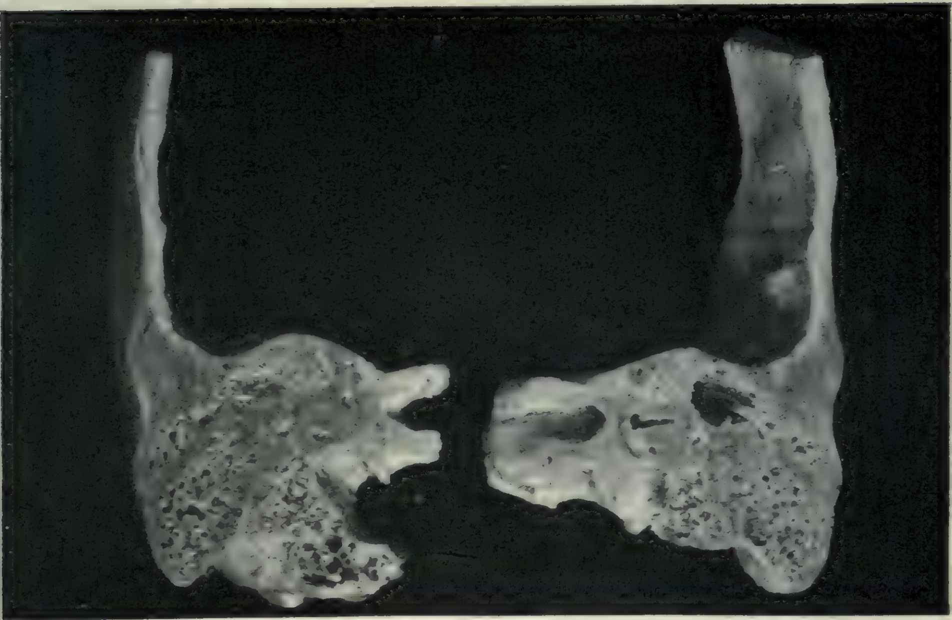
Right.—Outer antral wall partly dense and partly cellular. Fine cells in the up- er mastoid. Diploetic lower mastoid.



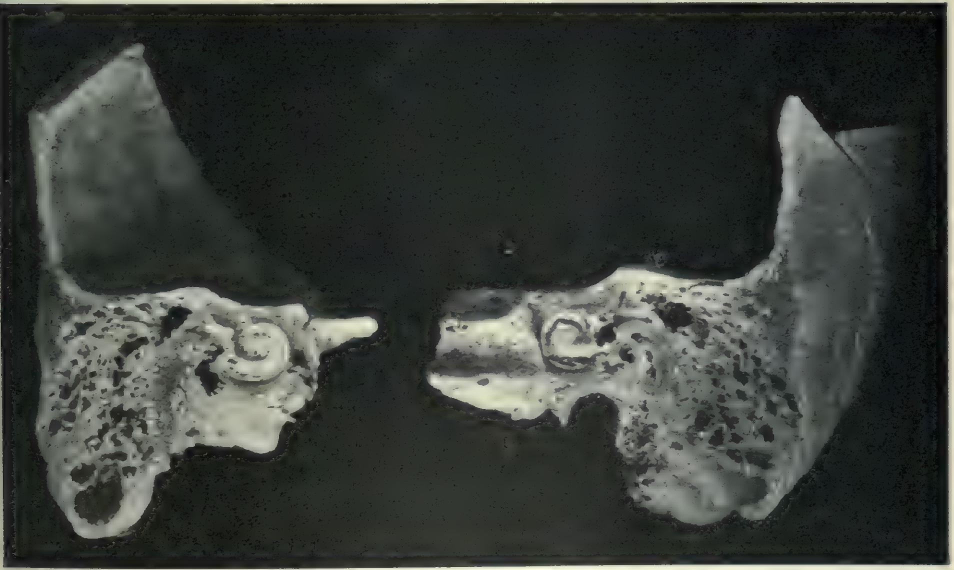
Left.—Diploetic infantile type.

No. 29.—FEMALE, AGED 61 YEARS.

Gross asymmetry.



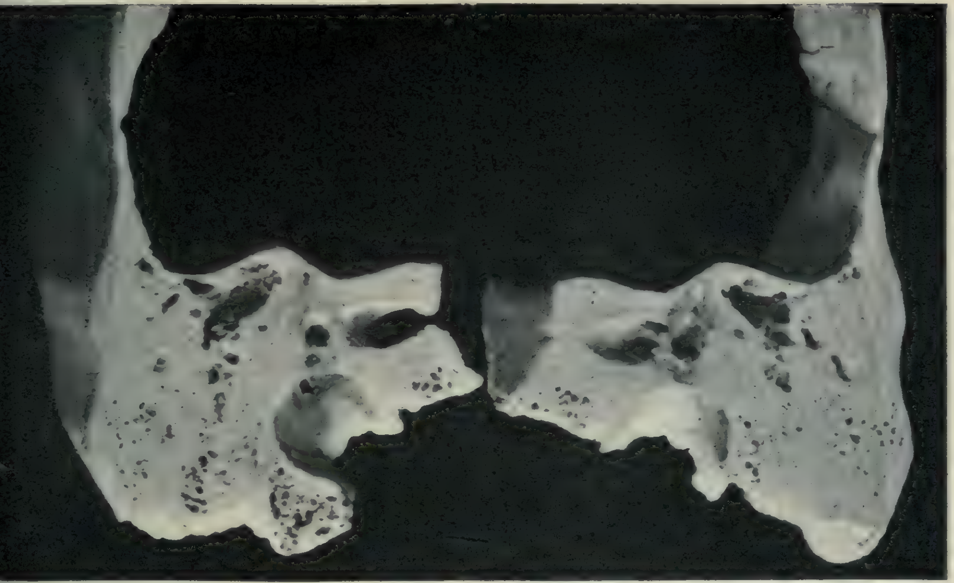
Right.—Diploetic infantile type. The squamous is plainly marked off from the petrous element of the mastoid.



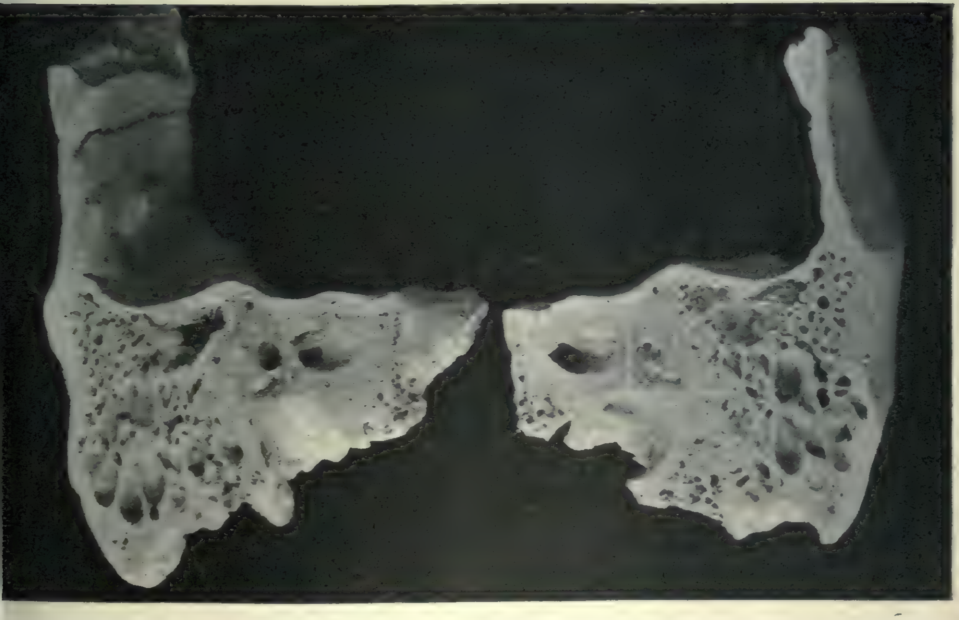
Left.—Cellular outer antral wall and entire mastoid. The elements are also well marked off.

No. 30.—FEMALE, AGED 73 YEARS.

Gross asymmetry.

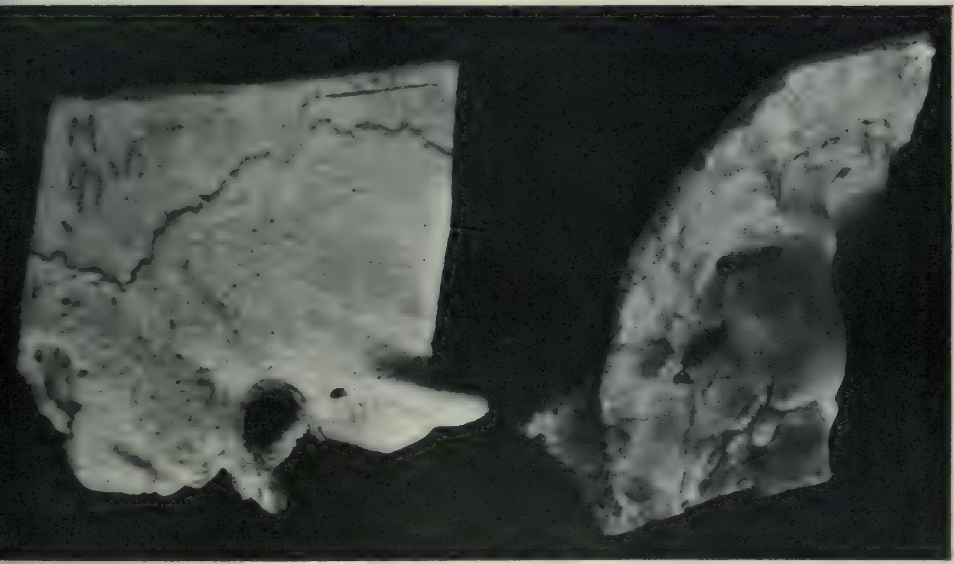


Right.—Diploetic infantile type. The diploe is very dense.



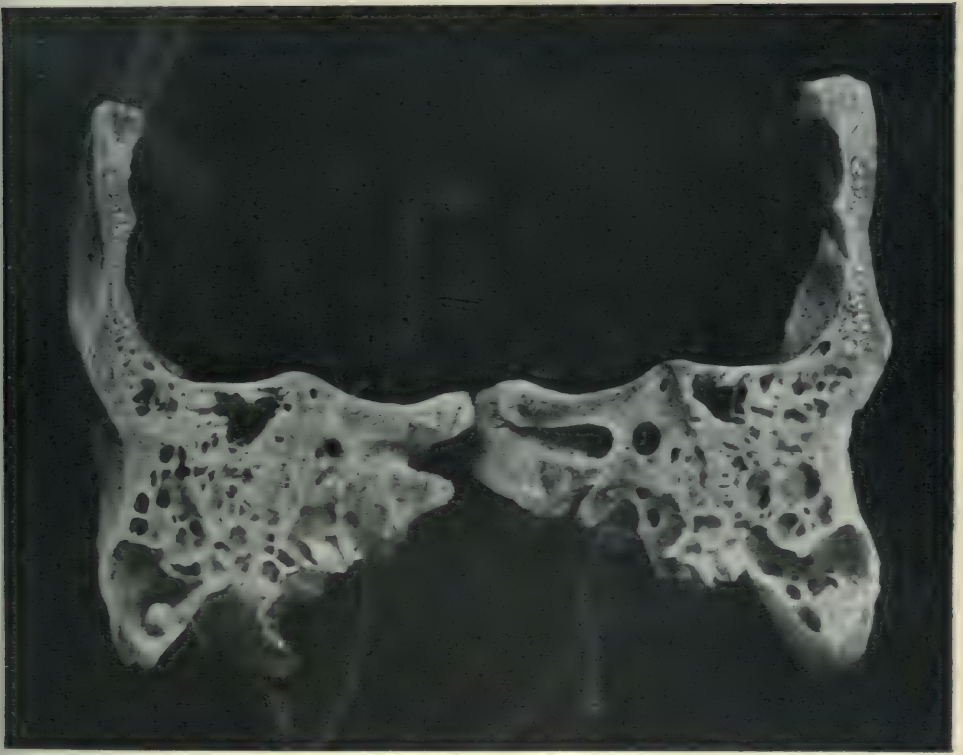
Left.—Outer antral wall and upper mastoid cellular. Lower mastoid diploetic.

No. 31.—MALE, AGED 9 YEARS.



Right Bone.—Showing an external opening for the petrosquamosal sinus in the base of the zygoma on the left fragment. The grooving along the inferior surface on the right fragment. The opening and grooving were absent on the left side.

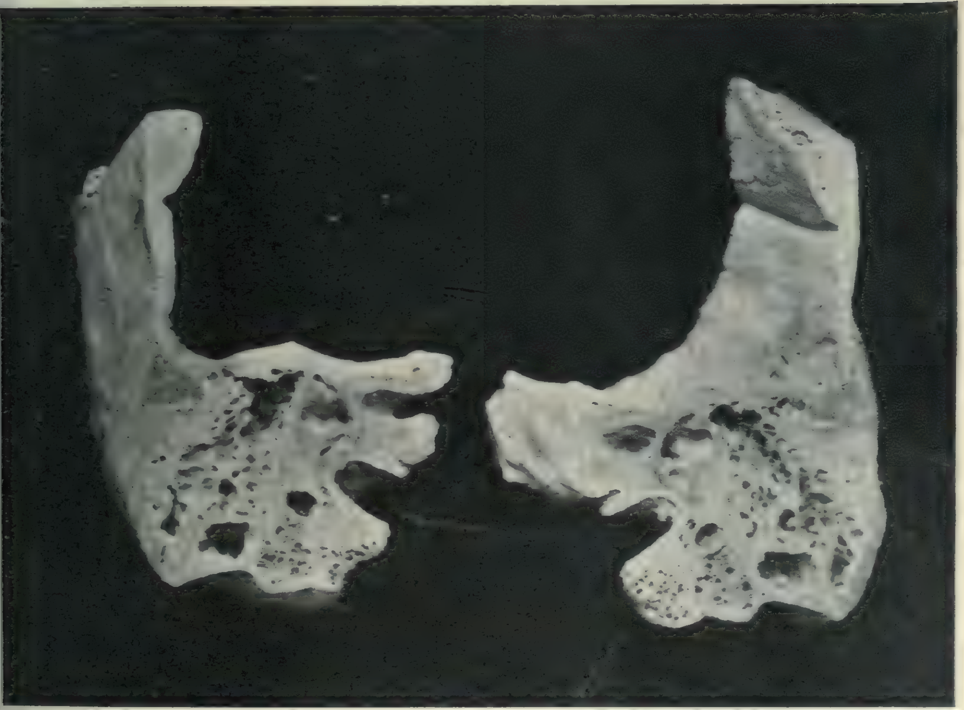
No. 32.—MALE. AGED 59 YEARS.



Right Bone.—A pedunculated exostosis is present, projecting from the inner wall of a large lower mastoid cell.

No. 33.—FEMALE. AGED 61 YEARS.

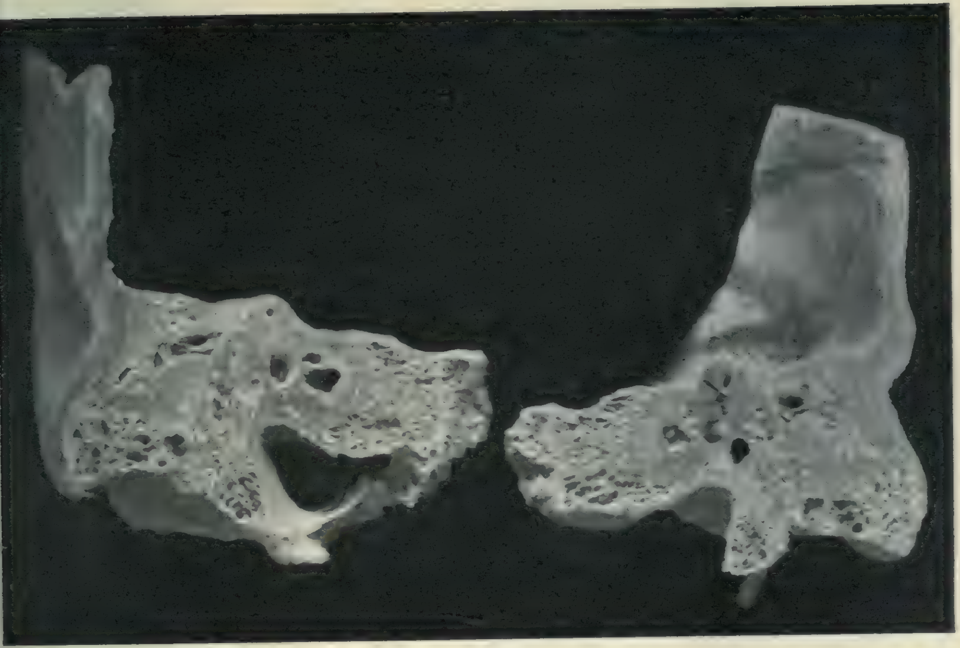
Malformation of the external bony semicircular canal.



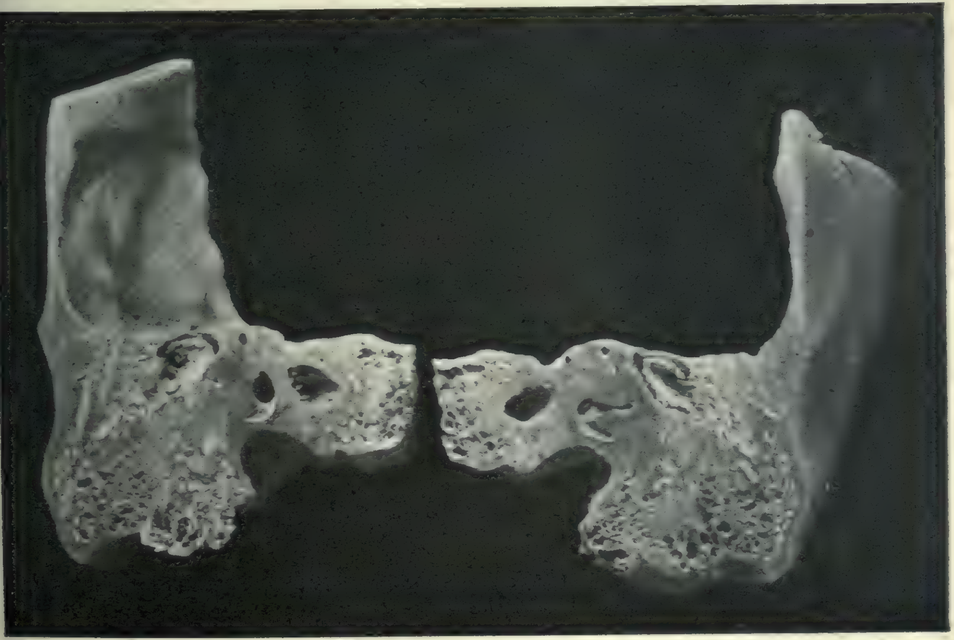
Right Bone.—The canal is represented by a wide cavity opening widely into the outer wall of the vestibule. A small nipple-shaped mass of bone projects from the back part of the sloping roof, and is the only attempt to make the ordinary partitions. The other parts of the labyrinth are normal, and so is the left labyrinth.

No. 34.—MALE, AGED 39 YEARS.

Symmetrical disease in symmetrical bones.

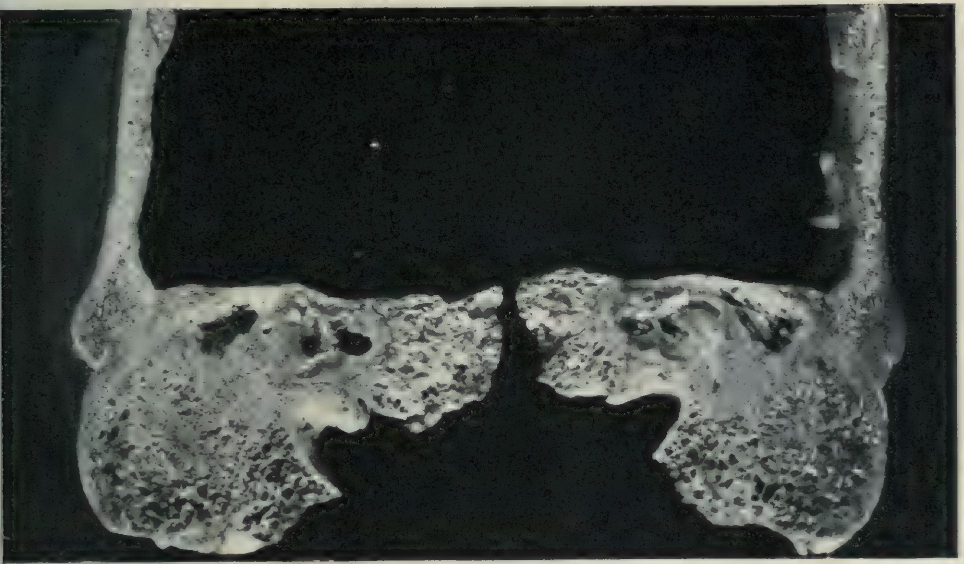


Right.—Chronic middle ear suppuration. Perforation in Shrapnell's membrane. Cholesteatoma filling the antrum. Diploetic infantile type.

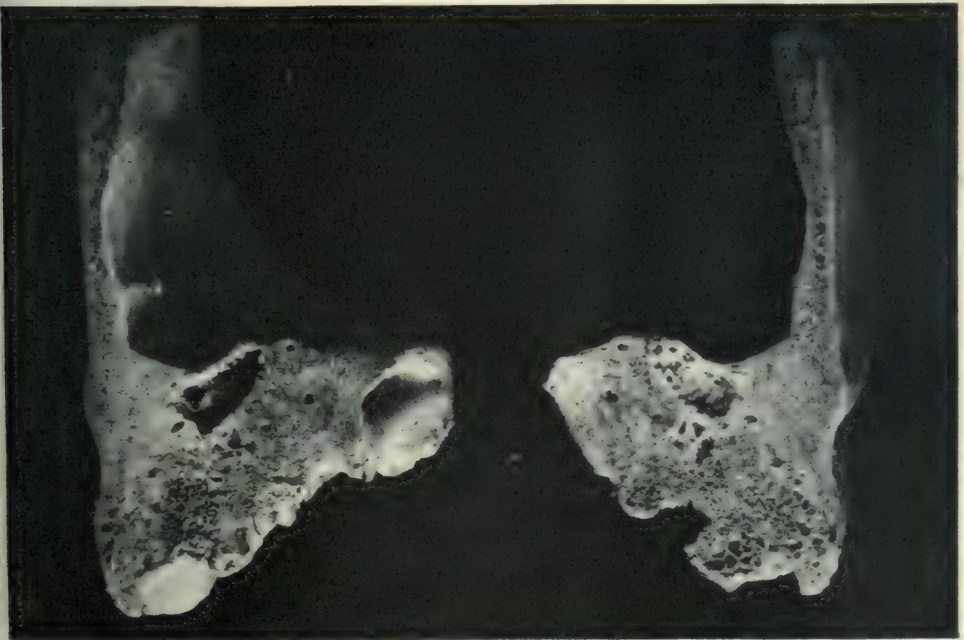


Left.—Symmetrical with the right in type and disease.

No. 35.—FEMALE, AGED 77 YEARS.

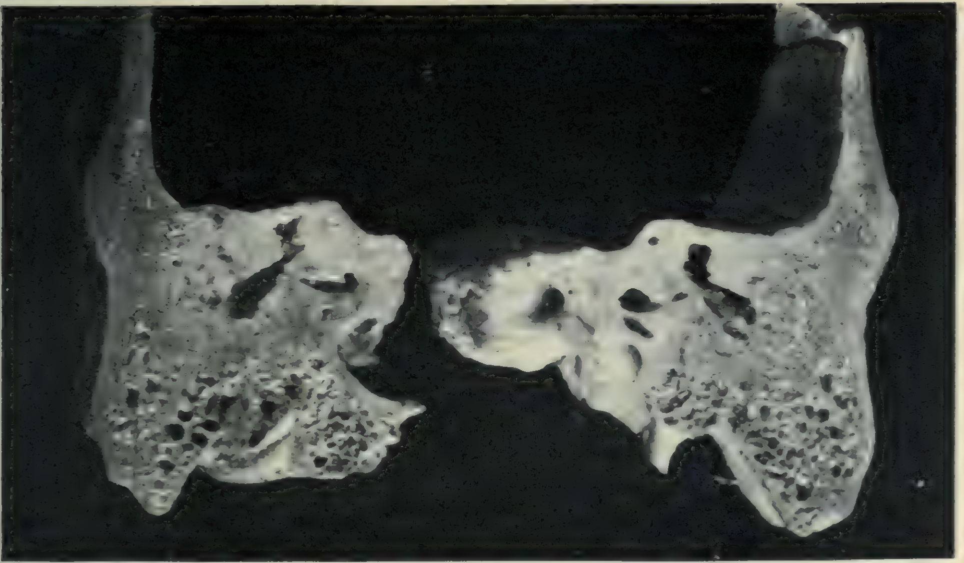


Right.—Diploetic infantile type. Chronic middle ear suppuration. Pus was present in the antrum. The fetal cells lining the outer antral wall are quite destroyed and the antral walls smoothed out. The aditus is much narrowed by osteosclerosis of the roof and fresh bone formation at the apex.

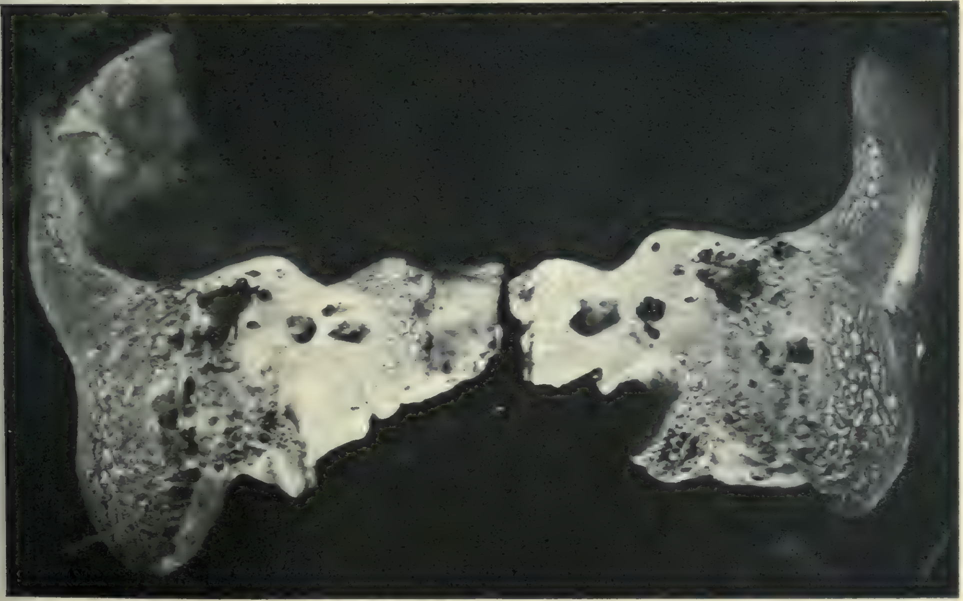


Left.—Diploetic infantile type normal. Intact membrane and ossicles. The middle fossa dips down, causing a sloping root to the antrum, a condition not present on the right side.

No. 36.—MALE, AGED 49 YEARS.



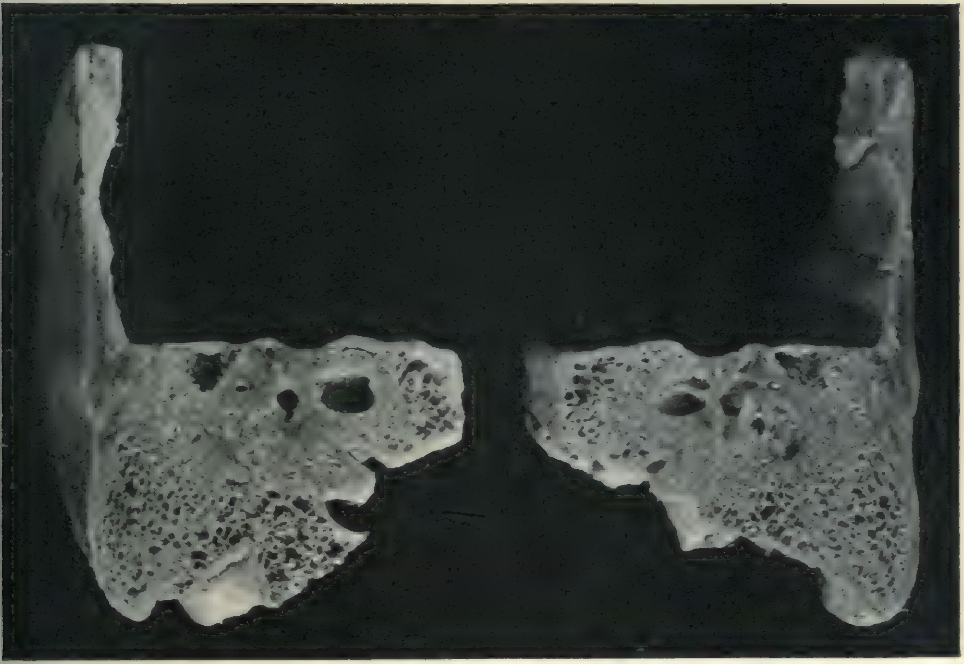
Right.—Diploetic infantile type. Chronic middle ear suppuration. Pus was present in the antrum, which is much narrowed by osteosclerosis of the fetal cells lining the outer antral wall, the osteosclerotic condition is well marked off from the normal dense outer antral wall which is usual in this type of bone. The antral walls are smoothed out.



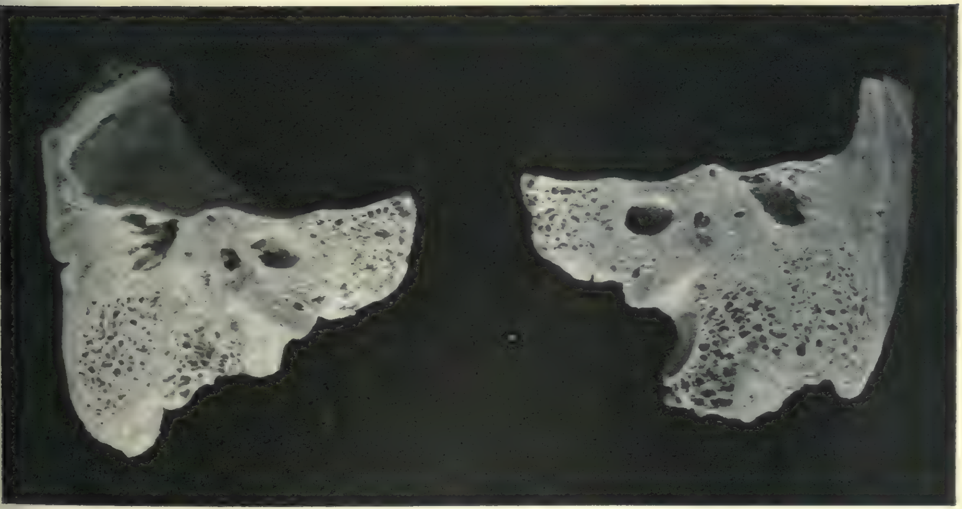
Left.—The outer antral wall is dense and the mastoid mostly diploetic, a few cells run downwards from the apex of the antrum. The membrane and ossicles are intact.

No. 37.—FEMALE, AGED 36 YEARS.

Fixation of the stapes.



Right.—Diploetic infantile type. Bony fixation of the stapes.



Left.—Symmetrical with the right in type and disease.

III.

REPORT OF A CASE OF BRONCHOSCOPY FOR MULTIPLE FOREIGN BODIES (ALMOND SHELL AND PULP) IN A CHILD TWO YEARS OF AGE, WITH SOME OBSERVATIONS UPON BRONCHOSCOPY IN INFANTS AND YOUNG CHILDREN.

By J. R. Winslow, B. A., M. D.,

Baltimore.

On January 5, 1911, at 2 p. m., in accordance with a telegraphic appointment, I met Dr. F. G. Wright, of Chambersburg, Pa., at the University Hospital, in consultation upon the case whose history follows:

Iona B., aged 2 years, was playing upon the floor two days previously, when her mother's attention was attracted by her crying, and she noticed that the child was blue in the face and breathing badly. She immediately held the child up by its feet, slapped her back and running her finger down the throat removed a large amount of almond shell and pulp; this resulted in greatly improved respiration, and the mother supposed that the nut had all been removed.

Since this time, however, the child has at times exhibited embarrassed respiration and occasionally cyanosis.

Examination.—Respiratory movements much shallower on left side and respiratory sounds lost below the second rib, no rales present; temperature, 98 2-5°; pulse, 118; respiration, 28. Slight supraclavicular retraction on left. Right lung normal.

The patient was admitted to the University Hospital and the larynx examined under cocain with the direct Jackson speculum. Nothing was seen in the glottic or subglottic space, and realizing the impossibility of passing a bronchoscope through a larynx of such size without undue force, tracheotomy was determined upon.

Meanwhile a radiograph was obtained, which, as was to be expected from the nature of the object (nut), revealed no foreign body, but only enlarged peribronchial glands.

On January 6, 1911, at 3 p. m., assisted by Dr. F. G. Wright, I performed a low tracheotomy, under chloroform anesthesia.

Immediately afterwards, with Dr. H. C. Davis in charge of the patient's head and the bronchoscopes, I passed a 7 mm. Jackson tube into the left lower lobe bronchus, which was systematically examined; the tube was, however, too large to enter the upper lobe bronchus. A considerable amount of milky pulp was found in the bronchus and removed with mops; no shell could be discovered.

I had expected to employ suction with Killian's aspirator, in the hope of aspirating the material from the smaller bronchus, but the patient became cyanotic, the pulse weak, and oxygen and amyl nitrite had to be administered.

The bronchoscopic examination was of necessity discontinued, having occupied about thirty minutes.

The tracheotomy wound was left open, long sutures were inserted in the lips, and the nurse instructed to pull the wound open should the patient cough.

The following day (January 7th) the temperature shot up to 103.4° F., respiration 140. Embarrassed respiration, diminished resonance with loss of inspiratory and expiratory murmur below the second rib were present on left side; tubular breathing was heard at left apex.

Subsequently marked cough with expectoration developed. In short, the patient manifested well marked pneumonia.

At this period Dr. C. W. McElfresh was called in consultation and placed in charge of the medical treatment of the case.

From the 9th to the 15th the patient went through the varying phases of a typical pneumonia.

On January 13th the tracheotomy tube, which had been inserted the day following the operation, was permanently removed and the patient was able to breathe through the mouth readily.

On January 17th, the thirteenth day, the patient was discharged from the hospital to return home; temperature 97.3°, pulse 110, respiration 24. Tracheotomy wound nearly closed, general condition much improved. The subsequent history of the case is derived from two letters sent by Dr. Wright. The first, dated February 16, 1911, reports:

Dear Doctor:—The child is still alive, but very ill. When she first came home she was very well, except a running ear and a solid patch a little larger than a silver dollar in the left apex. It seemed as though the trouble would subside and the foreign body become encapsulated, but she developed an influenza (every one here has it) and after a week of coughing developed a pneumonia involving at least the whole of the upper left lobe. After having the pneumonia one week the scar in the trachea opened spontaneously and I opened the skin, allowing a free discharge. The next day she coughed up an oval piece of almond kernel about 6x3 mm. That is four or five days ago, and she is slightly improving. If she gets well will send full data. WRIGHT.

An extract from the second letter, dated February 29, 1912, gives the final outcome of the case:

Dr. John Winslow.

Dear Doctor:—Today I examined Iona B. Sorry we did not report earlier, but they had moved. Have only lately located them. The child is apparently perfectly well, with no signs to show where the trouble in the chest was.

The scar in the neck is rather broad, but the trachea seems solid. After a slow convalescence she recovered fully.

Very truly yours,
FAIRFAX G. WRIGHT.

A more unfavorable case could scarcely be imagined than this one, presenting many difficulties.

1. The child's age (two years) and underdevelopment.
2. The nature of the foreign body, a pulpified nut, furnishing multiple particles which were spattered all over the lung surface, and doubtless entered every bronchiole.
3. The bronchus involved, the left, the most difficult to examine.

I have hesitated whether to call this a successful or an unsuccessful case; while technically I did not succeed in removing all of the foreign body by bronchoscopic methods, yet I am firmly convinced that had the nut-pulp not been removed from the main bronchus, tracheotomy alone would not have enabled the child to clear its lung and survive the first pneumonia.

From a life-saving standpoint the case was most successful, and one in whose outcome every one concerned has reason to feel gratified.

The pathologic condition in such a case is well illustrated in that reported by F. E. Hopkins (Trans. Am. Lar. Ass'n, 1911).

A female child, four years of age, inhaled a peanut; careful bronchoscopic examination at two sittings failed to reveal a foreign body and the child died on the second day.

"Autopsy showed many (24) small fragments of nuts scattered throughout the lungs and around each a pneumonic area.

"Instead of a single nut occluding the trachea or larger bronchi, the many fragments of the well-chewed nut were shot into the smaller bronchi."

Thomas Hubbard reports similar autopsy findings (ibid.) in a peanut case, in his practice.

Cases of foreign bodies in the lungs of young children and infants (say of four years and under) are coming under our care in increasing numbers, because through the writings of Killian, Jackson, Ingals, Coolidge, Halsted, Mosher, Hubbard and other masters of bronchoscopy, the general profession is being educated to recognize these conditions and their proper method of treatment.

These cases constitute the most difficult in the whole field of bronchoscopy, both on account of the small size of the respiratory passages at this period of life and the difficulties of instrumental manipulation, as well as the nature of the objects usually encountered.

While a young child is liable to place almost anything in the mouth and inhale it, owing to the small size of the glottis (6 mm. infants, Jackson—7 mm. three years, Killian) large objects cannot pass through into the lower passages, therefore many of the foreign bodies commonest in adults are rarely found in the lungs of very young children (pieces of bone), while those encountered belong to the class most difficult of removal, small or multiple objects (nut shells, pulp, beads, pins, beans, pebbles, etc.).

The younger the child the greater the difficulty and urgency, and the mortality is high, despite successful removal. So that it has seemed to me that these cases constitute a group of themselves well worthy of the discussion of this representative body, and for this purpose I have brought the subject before you.

What is our best course of procedure in this class of cases? Should we tracheotomize at once, as the primary procedure, or has the advent of bronchoscopy largely abolished the necessity for this operation?

The desirability of upper bronchoscopy (without tracheotomy) is obvious, and it should be the routine method were there no disadvantages associated with it.

In prebronchoscopic days tracheotomy was the method of choice, whose success is attested, among numerous others, by the remarkable series of four cases of foreign bodies in the bronchi of small children under two and one-half years of age, reported by our fellow T. H. Halsted (Trans. Am. L. R. O. Soc., 1902).

In these young children upper bronchoscopy has serious objections attached to it. The anatomic structures are small, rendering the manipulation of instruments difficult and resulting in loss of time and irritation of tissues.

A study of the cases reported shows that while upper bronchoscopy has been frequently attempted for the removal of foreign bodies in infants, in a large percentage, if not the majority of the cases, tracheotomy has become ultimately necessary for successful removal.

Now, if such be the situation, why not tracheotomize at once and operate by the easier and more certain route (lower bronchoscopy)?

In a recent article (*Deutsch. med. Wochenschrift,* June 29, 1911) G. Killian has made a most valuable contribution, in which he calls attention to another aspect of this subject, namely, the frequency with which tracheotomy or intubation becomes necessary after upper bronchoscopy, even when successful, reporting a series of nineteen cases under seven years of age, some of them personal and some derived from literature. He also cites a series of thirty-five cases reported by Schneider, of Moscow, of which five required intubation or tracheotomy. He concludes that these procedures stand in a causal relation to upper bronchoscopy, inasmuch as the changes necessitating them occur within a relatively short period (6 to 37 hours) afterward; that the site of the change is the subglottic space, as evidenced by the stridor and the results of intubation or tracheotomy, and in a few cases by direct or indirect laryngeal examination.

We know from both clinical experience and postmortem evidence that inflammatory swellings are prone to occur in the subglottic space. Children from the seventh, and especially from the fourth, year downwards are especially liable to such swellings after upper bronchoscopy. In this connection the relation of the caliber of the tube to the width of the subglottic space becomes of paramount importance. No reliable measurements have hitherto existed. In order to furnish such data, Killian undertook the measurement of twenty-two children's larynges in fresh cadavers. For this purpose the employed a series of semicircular metal sounds of increasing size, observing which one passed the cricoid region easily, which with difficulty, which not at all. In this way he determined that the width of the subglottic space stands in no especial relation to the sex of the child, and in but little to the age, the most important and hitherto neglected moment being the bodily development.

Upon this basis he has constructed the following table:

Body length.	Diameter of subglottic space measured with calibrated metal sounds.	Calculated periphery of the lumen.	Age.
45 cm.	3.5 mm. (4 not)	10.99 mm.	8 mos.
50 “	4.5 mm. (5 not)	14.13 “	4 “
52 “	5 mm.	15.7 “	3.5 “
53 “	4.5 mm. (5 not)	14.13 “	3 “
53 “	5.5 mm.	17.27 “	newborn
53 “	4.5 mm. (5 not)	14.13 “	“ “
56 “	4.75 mm.	14.91 “	?
58 “	5 mm. (5.5 pressure)	15.7 “	2.5 mos.
58 “	5.5 mm.	17.27 “	4 “
60 “	5.5 mm.	17.27 “	9.5 “
62 “	5.5 mm. (6 with difficulty)	17.27 “	6 “
62 “	6 mm. (6.5 not)	18.84 “	18 “
63 “	5.5 mm.	17.27 “	9 “
64 “	5.5 mm.	17.27 “	13 “
65 “	6.5 mm. (7 not)	20.41 “	8 “
70 “	5 mm.	15.7 “	16 “
80 “	6.5 mm. (scarcely)	20.41 “	12 “
80 “	6.5 mm. (7 not)	20.41 “	24 “
86 “	7.5 mm. (8 not)	23.44 “	48 “
94 “	7 mm. (7.5 not)	21.98 “	36 “
120 “	8 mm.	25.12 “	7 yrs.
? “	9.5 mm.	29.83 “	10 “

A study of this table shows that the subglottic space is much narrower than is commonly supposed; that it increases gradually with the body length, but not commensurately, and is subject to great variations. Comparison of these measurements with the age of the child, in the series collected, shows that in some of the cases the bronchoscopic tube was too large for the subglottic space, and must have caused trauma.

Killian observes that we still require a great deal of information as to the anatomic relations of the child's larynx at different ages and different grades of bodily development, and promises to take this up for future research.

The selection of method must be individual, depending upon a number of considerations.

1. Age of the patient: as Killian has demonstrated, this is more a matter of physical development than of age: nationality also may have a bearing. At least my intubation experiences have shown me that in certain nationalities (Italian) the larynx is smaller than the corresponding age.

Some years ago Ingals announced that he had rarely found upper bronchoscopy satisfactory in children under three years of age.

Recently Finder, apropos to an unsuccessful case of a piece of bone in the right bronchus of an eleven months old child, reported to the Berlin Laryngological Society, stated that henceforth he will resort to inferior bronchoscopy in all children in the first year of life.

In discussing this case E. Mayer went still further and considers inferior bronchoscopy preferable in children six years of age and under. Brünings advises it in all children under two years of age, as a routine procedure.

Nehrkorn recommends low bronchoscopy in all young children.

Jackson, however, regards tracheotomy as being "unnecessary nine times out of ten, and believes that it should be limited to dyspneic cases." I should like to know whether he intends this to apply to these very young children.

2. The nature of the foreign body is of great importance in determining this question.

Objects which are liable to swell so that they cannot be withdrawn through the subglottic space (beans) should be removed by the lower route (Nehrkorn, Killian).

Likewise brittle objects which are liable to be broken into several fragments, or multiple objects necessitating a great deal of manipulation; objects which are irritating in themselves and certain to be followed by pulmonary inflammation (peanut shells, pepper corns), should be removed without irritation of the subglottic space by instruments.

3. The duration of the condition is of a determining moment: the presence of a foreign body rapidly produces a catarrhal condition of the air passages in children, increasing the vulnerability of the mucous membrane to instrumental manipulation; therefore, when the foreign body has remained for a long time, tracheotomy is indicated (E. Mayer, Killian).

4. The side affected must be considered, for it is much more difficult to remove a foreign body from the left bronchus, especially by upper bronchoscopy. Among thirteen cases in children collected by Killian, in nearly two-thirds the foreign body was found in the left bronchus.

5. The condition of the subglottic space is of paramount importance: this should always be determined by direct or indirect laryngeal examination before undertaking upper bronchoscopy.

Any outspoken subglottic swelling constitutes a contraindication to upper bronchoscopy.

The passage of the bronchoscopic tube will inevitably result in the necessity for tracheotomy; it is therefore better to perform this operation primarily and employ lower bronchoscopy.

In conclusion, it seems to me that the present situation with regard to foreign bodies in the lungs of young children has been well summarized in the advice of Hubbard. "When in doubt do tracheotomy."

REFERENCES.

Halsted, T. H.: Trans. Am. Lar., Rhin. and Otol. Soc., 1902.
Killian, G.: Trans. Am. Lar., Rhin. and Otol. Soc., 1907.
Killian, G.: Deutsch med. Woch., June 29, 1911.
Brünings, W.: Zeitschr. für Ohrenheilk., Dec., 1910.
Finder: Verhandl. der Laryng. Gesellsch. zu Berlin, 1911.
Mayer, E.: Verhandl. der Laryng. Gesellsch. zu Berlin, 1911.
Hubbard, Thos.: Trans. Am. Laryng. Ass'n, 1911.
Jackson, Chevalier: Trans. Am. Laryng. Ass'n, 1911 (discussion).
Hopkins, F. E.: Trans. Am. Laryng. Ass'n, 1911.

IV.

TUMORS OF THE LARYNX.*

By Robert Levy, M. D.,

Denver.

It is not the purpose of this paper to present an exhaustive treatise upon laryngeal tumors, but rather to call attention to a number of practical points in the operative treatment and to emphasize these points by illustrative cases.

Although intralaryngeal tumors were probably recognized and removed prior to the advent of the laryngoscope, it was not until this instrument came into use that their recognition or removal was attended with any degree of accuracy. Within a few years of Garcia's discovery the first operation for laryngeal neoplasm was performed. The question of priority, as in many other subjects, is a vexed one. Wright gives Lewin credit for the first intralaryngeal operation for the removal of a laryngeal growth under the guidance of the mirror, which he is said to have performed on July 20, 1860, while Jurasz credits Victor von Bruns with the first operation, performed in 1861, and does not even mention Lewin. From these dates the number of tumors observed and extirpated by this method grew with great rapidity, and the scarcity of the growths of recent years has been ascribed to the avidity with which they were removed in the early days of laryngoscopy. As Wright so well expresses it, "no subject so immediately engaged attention as that of laryngeal tumors, forming striking pictures in the laryngeal mirror, causing marked and distressing symptoms, capable of immediate relief by means of instruments under the guidance of the laryngoscope, and last, but by no means least, affording the operator a chance to display in the most brilliant manner his newly acquired skill."

The removal of tumors from the larynx per vias naturales and under the guidance of the laryngoscope may be termed the laryngoscopic method, to distinguish it from more recent intralaryngeal manipulation by means of endoscopic instruments.

---

*Read at the annual meeting of the Colorado State Medical Society, September 24-26, 1912.

For the successful laryngoscopic operation proper illumination is absolutely essential. The source of light must be brilliant, white, uniform and free from conflicting shadows. These qualities are best obtained by using the Nernst electric light placed in a suitably constructed metal cylinder.

The instrument table should be conveniently placed to the operator's left, and should contain, besides the instruments needed, gauze sponges, sterile napkins, spirit lamp, etc. Three assistants are desirable. The duties of the first assistant are most important, and consist in holding the patient's tongue, if it is found that the patient cannot do this himself, in controlling the epiglottis and in manipulating intralaryngeal instruments under the direction of the operator, should it be found necessary to use more than the one in the hand of operator himself. A second assistant presides over the instrument table, and a third holds the patient's head and retains the mouth gag in position if it becomes necessary to use one. At times it is necessary to pass a silk ligature through the tip of the tongue, instead of holding the tongue with a napkin. This is particularly serviceable in operating under general anesthesia.

One of the greatest obstacles to satisfactory endolaryngeal operation is a depressed epiglottis, especially under general anesthesia. To control this the device described by Horsford, in which a properly constructed needle holder enables one to pass a ligature through the free border of the epiglottis, is recommended. This ligature can then be held by the first assistant or grasped by a pair of hemostat forceps and allowed to hang out of the mouth. Pfau's modification of Horsford's instrument (Figure 1) makes it easier of application in many cases. By its use a needle properly threaded is placed in one of its blades, which, when closed upon the opposite blade, permits the needle to be grasped and withdrawn with the instrument, the ligature remaining in the tissue.

An additional aid to laryngoscopic operation, as indeed it is to careful examinations, is the magnifying anastigmatic mirror of Brüning, which enlarges the laryngeal picture two and one-quarter times.

Anesthesia.—Endolaryngeal operations may be conducted under local or general anesthesia.

Local Anesthesia.—Since the advent of cocain, operations within the larynx have become greatly simplified, and many of the previous difficulties in the way of preliminary training have been almost completely overcome. The proper local application of cocain or some of its substitutes is productive of uniformly satisfactory anesthesia.

Cocain remains the most reliable drug, but owing to its toxic properties, betaeucain may be substituted at times. Where unsatisfactory results are obtained one can usually determine a faulty technic in its application. In a few rare instances the use of this local anesthetic in quantity approaching the danger line has failed to anesthetize the mucous membrane of the larynx, so that the slightest touch of the instrument has resulted in a violent exhibition of the laryngeal reflex. There is a distinct difference between the retention of sensation and the reflex contraction of muscles. In these cases local anesthesia may fail.

Cocain poisoning is less likely to occur if it is so applied that little if any is swallowed by the patient. Its application directly to the field of operation is therefore desirable. Reflex contraction may be avoided by applying the anesthetic to the soft palate, posterior pharyngeal wall and posterior surface of the epiglottis. This being a large surface to cover, a solution may be sprayed upon the parts as a preliminary procedure. If cocain is used, it should not be more than a 2 per cent solution. The immediate effect of this is to cause, in addition to anesthesia, a most disagreeable and, to some individuals, alarming sensation of constriction. The dangers of poisoning are much greater where solutions are used in spray than where the remedy is applied by cotton applicator. The addition of a solution of one of the adrenalin preparations in the strength of 1/8000 will inhibit the cocain intoxication. This may be almost completely avoided, however, by substituting a 2 per cent to 4 per cent solution of betaeucain. It has been shown that this drug is very much less toxic than cocain, and while its anesthetic properties may not be so pronounced or reliable, it generally answers for the preliminary spraying. After waiting five minutes the field of operation should be carefully painted with a 20 per cent solution of cocain by means of the cotton applicator. This should be thoroughly moistened with the solution.

but not to the extent of permitting any of it to drip from the cotton. Repeated applications of the stronger solution should be made at intervals of two minutes until one determines by touching the parts that anesthesia is complete. The sensation of the patient of local constriction may also be a guide.

In a certain proportion of cases the application by means of the cotton swab is ineffectual. Here five drops of the 20 per cent solution may be applied directly to the field by means of the laryngeal syringe. This should, of course, be preceded by the preliminary spraying with the weak solution.

The submucous injection of cocain solution for anesthesia has been practiced by Herying and others. This is particularly recommended for those cases in which swollen or infiltrated masses are to be extirpated, such as are to be found in tuberculosis. The technic of this method is greatly facilitated by the use of Chappel's syringe. (Figure 2.) Having produced preliminary anesthesia, the needle of this instrument is pressed well into the submucous tissue and by a slight movement of the thumb the spring releases the piston, forcing the fluid into the parts. The advantage of this instrument is in the accuracy with which it can be used. Anesthesia by this method is more rapidly produced and persists for a somewhat longer time than by the method of topical application.

The method of inducing local anesthesia by injection into the superior laryngeal nerve has been described by Frey and others, and recommended especially for the relief of painful affections of the larynx. It may, however, be satisfactorily utilized in the intralaryngeal operations, especially in excessive irritability of the upper half of the larynx. Twenty drops of a 1 per cent solution of cocain in combination with 1/1000 solution of adrenalin gives satisfactory results. The success of this method depends upon waiting the proper length of time after the injection before beginning intralaryngeal manipulation. In some cases anesthesia is complete in three minutes; in others, twenty minutes must be allowed to elapse.

General Anesthesia.—In relatively few instances does it become necessary to administer a general anesthetic for endolaryngeal operations, except in cases of children or young adults. In the author's experience the adolescent age is particularly resistant to satisfactory local anesthesia of the larynx.

When general anesthesia is necessary, the choice of anesthetic becomes a question of considerable importance. Generally speaking, ether is to be preferred, although its well known irritating effect upon the mucous membrane of the upper air passages, producing excessive secretion, is a decided disadvantage. On the other hand, the element of safety enters as a more important factor, and particularly where the almost erect or sitting position of the patient becomes a necessity. Chloroform, while producing ideal anesthesia and specially useful in children, adds greatly to the dangers and compels one to keep the patient in a reclining or semirecumbent position, a most difficult and at times impossible position for intralaryngeal operations.

The excessive secretion in ether anesthesia may be greatly diminished by the administration of atropin one hour before the operation.

In using general anesthesia the patient should be placed in an operating chair so constructed that he may be raised to a sitting posture or lowered to the recumbent position at will. Anesthesia should be begun in the recumbent position, and when complete the patient should be gradually raised to the desired position. A chair is preferable to an operating table, in that it permits the operator to sit directly in front of the patient, and not to either side, which places him at an inconvenient angle for ease and accuracy of manipulation. In the case of young children the anesthetic may be begun upon a table, the patient being lifted to the lap of a nurse before commencing the operation.

Complete surgical anesthesia is absolutely essential. It is extremely difficult to continue the administration of the anesthetic during the operation. This may be partially accomplished either by holding a gauze sponge saturated with the anesthetic and grasped by a long pair of forceps in the vicinity of the nose and mouth, or by the use of a properly constructed tube, similar to that of the Junker inhaler, placed in the right angle of the mouth. As a rule, this will not interfere with the use of the laryngoscope, and is out of the way of laryngeal forceps or other operating instruments.

It is well known that the laryngeal reflex is one of the last to disappear under anesthesia. Even when profound narcosis has been established, the moment an instrument is introduced into the larynx, reflex spasm occurs. In order to overcome this, a solution of cocain and adrenalin should be applied, especially over the posterior surface of the epiglottis. This can best be done by means of the cotton applicator. The strength of the solution should vary with the age of the child. Young children do not bear cocain well. A 1 per cent to 2 per cent solution in combination with a 1-10,000 adrenalin solution, answers every purpose. In young adults stronger solutions may be used, generally 5 per cent being sufficient. If this does not answer, the percentage may be increased to 15 per cent or 20 per cent, care being observed to have the cotton moist but not dripping.

Before proceeding with the description of operative methods, a brief consideration of the relative value of endolaryngeal and external operations may not be out of place.

Although the laryngoscopic method is less applicable to carcinoma, even here it may be of value. It has been advocated by Gouguenheim and Lombard:

First—In certain varieties of intrinsic cancer which remain limited for a long time, showing no tendency to spread.

Second—In old people whose advanced age would make us hesitate before proposing a large surgical intervention, and

Third—In certain pedunculated epitheliomata, the rapid removal of which is necessary to prevent suffocation.

In malignant disease the most generally useful purpose, after all, to which intralaryngeal surgery may be put is for that of diagnosis. The operation of removing a section may be performed with impunity in all cases. The caution so frequently given, that unless the patient be prepared for a radical operation the growth should not be attacked, is only of minor importance. It is absolutely essential to confirm the clinical diagnosis by histologic examination. If this examination proves negative, no harm has been done, and the way is clear for further and possibly successful treatment. If this examination proves positive, the growth may or may not take on rapid growth. Should the patient refuse more radical operation and the tumor rapidly progress, the fatal termination may be somewhat advanced; this, however, must be looked upon as of much less importance than the question of an early confirmed diagnosis, which must remain, as previously stated, the saving factor in a possible cure. If the malignant growth has already assumed large proportions and has invaded surrounding glands, the clinical diagnosis usually suffices. The tumor may have been of very slow growth, and it is much wiser to leave it alone rather than to encourage its growth by removing a section to confirm a diagnosis which needs no confirmation. What has been said above with regard to removing a piece for diagnostic purposes, applies only to small circumscribed tumors of doubtful diagnosis.

The two varieties of malignant growths found in the larynx, as elsewhere, are sarcoma and carcinoma. For many years the relative value of endolaryngeal and extralaryngeal operations for these neoplasms has been a matter of much discussion. the burden of which has had reference to carcinoma. Sarcoma has been given comparatively little consideration, evidently due to the fact that it is of relatively rare occurrence. The fact that sarcoma is less likely to spread to adjacent structures, especially in its early stages remaining circumscribed and confined, favors the endolaryngeal method somewhat more in this variety of malignant growth than in carcinoma. Laryngologists have from the beginning leaned naturally to the laryngoscopic method. Unfavorable results, however, have compelled the conclusion that in but few instances is it possible to completely eradicate a malignant growth by removing it through the natural passages. A few brilliant results have been reported, especially by Fränkel, who among others has been the most ardent advocate of this method. Statistics have been of comparatively little value, for, as Jurasz has pointed out, the value of statistics depends upon more careful differentiation of the period or state of development of the growth at which the operation is undertaken. Cases of cure by both methods have been reported, the early diagnosis being in all instances the important factor. The improved technic of today renders it possible to obtain results by radical external operations which were impossible a few years ago. At that time, while it was possible to completely extirpate a growth by radical measures, the gravity of the operation was so great that the attempt was generally made to remove the mass by simpler procedures. Today, however, less is to be feared from the operation than from the rapid development and extension of the malignant growth, and therefore those cases which were formerly attacked by endolaryngeal methods, in the hope of avoiding more serious operation, and were followed by indifferent results, are today more generally and more successfully removed by laryngectomy or laryngotomy.

Nevertheless, endolaryngeal procedures in malignant growths may find their uses.

Generally speaking, all benign tumors should be removed through the natural passages rather than by external incision. The size and position of the tumor are the most important determining factors in its removal. Supraglottic tumors, infraglottic tumors, providing they do not extend too far downward, and even intratracheal growths, may be extirpated under the guidance of the laryngoscope. The question of choosing between this method and the direct endoscopic procedure is not as yet capable of decision. Owing to the large number of laryngologists trained in the use of the mirror, compared to the few whose technical skill enables them to use easily the direct method, the former will doubtless for many years be the method of choice. It must be conceded, also, that patients are more readily trained to the required manipulation of the laryngoscopic method than they are to the method by the direct laryngeal speculum, which offers another reason for adopting the former. On the other hand, it is selfevident that owing to the situation of a tumor, it may be inaccessible except by direct laryngoscopy. This is peculiarly true of growths situated low in the larynx or in the trachea. The trained laryngologist may proceed with safety by first attempting the laryngoscopic method, failing in which he may then adopt the direct method.

In children, multiple papillomata are fairly frequent. Here the removal of these growths by the endoscopic method is often the procedure of choice. As a rule, whatever operation is undertaken in children, general anesthesia is necessary. The laryngeal speculum of Mosher and the tracheoscopic and bronchoscopic tubes of Killian and Jackson have made the removal of papillomata in children comparatively simple. For the removal of these tumors small, straight, cutting forceps or the spiral instrument devised by Mosher is recommended.

A method of suspension laryngoscopy recently described by Killian, although requiring elaborate apparatus, will probably add materially to the ease of intralaryngeal manipulations by the direct method. By the use of this suspension apparatus the operator brings the laryngeal cavity into the field of vision, holding it there, and leaving his two hands free for other manipulations.

Among other recognized measures for the removal of papillomata in children, it should be remembered that tracheotomy and intubation have both their advocates. It is a well recognized fact that no matter what operation or how frequently it is performed, papillomata recur with exasperating rapidity. It is also well recognized that these tumors have a life history, and that after a certain length of time they not only cease their growth, but retrogress. In undertaking operations for their removal, therefore, one is fortunate if he chooses the psychologic moment, that is to say, the particular time when the tumor has reached its maturity. In children the presence of papillomatous growths frequently causes alarming dyspnea. Tracheotomy performed at this time places the larynx at rest, following which the natural tendency for retrogression of these tumors asserts itself and their spontaneous disappearance frequently results. In one of the author's cases treated in this manner, a tracheotomy tube was worn for over two years. Various intralaryngeal manipulations had been unsuccessfully tried. The case made a complete recovery, after all attempts at removing the tumors had been discontinued, recovering with a good voice, by spontaneous disappearance of the growths.

In a certain proportion of cases in children in which the tumors do not develop at the entrance of the larynx, but confine their growth to the vocal bands and surrounding structures, intubation has been successfully used. Not only does the intubation relieve dyspnea, thus placing the larynx at rest, but the intubation tube causes a certain amount of gentle and continuous pressure which may have a beneficial effect. At any rate, the writer has successfully treated cases in this way, the last one being a child three years of age, in whom the intubation tube was worn almost continuously from November 7, 1910, to December 8, 1911, eleven months, with the exception of about four months, during which time the breathing was good. Becoming worse after a severe cold, reintubation was necessary. The child has now been without a tube about ten months, and when last heard from was breathing well and had had but slightly husky voice.

It is the author's practice to remove the tube at intervals of from three to six weeks, cleanse it and reintubate immediately, if necessary, or within a very short time. In some cases calcareous deposits form around the metallic tube which become a source of irritation and even cause superficial ulcerations. A new tube must be used whenever the one removed shows any sign of roughness.

The treatment of multiple papillomata of the larynx in children by fulguration, although not as yet found in medical literature, has been practiced by Dr. Harmon Smith of New York, and probably others. Dr. Smith's experience has been more extensive than other operators. In a personal communication dated September 19th, Dr. Smith states, "that fulguration will cut down these growths, but they soon return, and at the present writing I am unable to say that it has any features of a definite character beyond the fact that it removes the growths easily and prevents the necessity of tracheotomy." Dr. Smith's method is to apply the spark for a few seconds until the surface becomes white. The operation is performed under chloroform and cocain anesthesia and with the Jackson tube.

Besides the methods already mentioned, which are, of course, performed without the use of the laryngoscope, those under the mirror's guidance are by means of chemical caustics, galvanocautery, electrolysis, the sponge method of Voltolini, incision, forceps, the cold or galvanocautery snare and the guillotine.

Chemical caustics may be used in small nodules, small papillomata, pachydermia and tuberculous or syphilitic excrescences. They are of value in that form of multiple papillomata in which the neoplasm seems to take on the nature of diffused papillomatous infiltration. This condition is well illustrated in Figure 3. In this patient a large number of papillomata were removed by the forceps until it was found difficult to remove more than very minute sections. Here repeated cauterization proved beneficial. The best method of using chemical caustics is by fusing a crystal of nitrate of silver or chromic acid upon a suitably curved applicator, or inserting a crystal of chromic acid in the fenestrum of a hollow applicator, such as is used for carrying trichloracetic acid. (Figure 4.)

The galvanocautery is effective where delicacy of application is desired. It is particularly useful for the removal of small vascular growths and varicosities. After the removal of angiomata there frequently remains a small vascular point, which may be the beginning of a new growth, and which is best removed by the galvanocautery point.

Electrolysis is applicable to tumors with broad sessile bases as well as to those in which hemorrhage is liable to be an important factor. Fibromata, cystomata, chondromata and certain angiomata may be attacked in this manner. In some instances malignant tumors have been known to diminish in size by the use of electrolysis. This at best can only be a makeshift.

Figure 5 illustrates a variety of tumor readily treated by incision. The tumor proved to be, as its appearance indicated, a cystoma. Cystomata are nearly always broad at their attachments, and in many instances it is impossible to grasp them by means of the ordinary forceps in use. One of the numerous laryngeal knives, especially that devised by Herying, may be used. In this case incision evacuated a small quantity of thick opaque fluid. The sac disappeared and there was no recurrence.

The removal of tumors by laryngeal forceps is the most generally applicable of all methods. Many varieties of these instruments have been devised, and tumors have been removed by evulsion, by crushing and by abscision. It is generally conceded that cutting forceps are the most desirable, their action being more precise as well as more easily controlled. They should be so constructed that all portions of the larynx may be readily reached by permitting movements from below upwards as well as anterposterior and lateral. These qualities may be found in the Mackenzie or Scheinmann instruments. Among the best of the cutting forceps are those known as Cordes, which have also been called double curettes. Their action is from below upwards, and may be applied with great delicacy to almost any position in the larynx. Figures 6, 7, 8 show a fibroangioma which was readily grasped by a Mackenzie forceps and removed by evulsion. The bleeding that followed was somewhat more profuse than ordinarily, but was not alarming. The action of the Cordes forceps is well illustrated by Figures 9, 10, 11, showing a fibroma of the left vocal band.

The galvanocautery snare is particularly applicable to bleeding tumors; it also may be used for removing portions of neoplasms which cannot be removed in toto.

All tumors, of whatever nature, presenting any degree of pedunculated attachments, may be readily removed by the cold wire snare. Even tumors that have a sessile base may be attacked in this way, providing the projecting portion of the neoplasm is not smaller than the base.

Tumors springing from the epiglottis are readily surrounded by the snare wire. The most frequent variety of tumors found here are the lipoma and the cystoma. The snare is much more applicable to the former than to the latter. In applying the snare to tumors in this region, the straight variety used for operation within the nose, or that possessing a slight curve, may be easily applied. The method of procedure is slightly modified over that used for ordinary intralaryngeal operations. The modification consists in depressing the tongue instead of drawing it forward. For this purpose a tongue depressor with a long handle is desirable, the patient being instructed to hold the instrument in position himself. The laryngoscopic mirror in such case may be entirely discarded.

Tumors situated below the epiglottis, whether large or small, may be reached by the snare without much difficulty, providing they are slightly pedunculated. Among the numerous instruments devised for this purpose, the Schroetter snare, as improved by Coakley, is one of the best. The wire used should be sufficiently firm to remain in whatever position it is placed. It should be of fair strength, although this is of less importance than in other snare operations, such as are performed in the nose or for the removal of tonsils. Number 6 piano wire is small, strong and sufficiently stiff for all purposes. It should be threaded to the snare in such a manner that the loop rests in an anteroposterior direction for tumors situated in the anterior or posterior portions of the larynx. By introducing the ends of the wire into the eyes of the stylet from opposite sides, or both ends from the same side, one may obtain the two positions of the loop as desired. The loop should be large enough to readily surround the neoplasm. It should not, however, be too large, in which case it becomes difficult to enter the larynx with it, and also for the reason that the manipulation of drawing the wire tightly over the growth is thus prolonged.

In removing a tumor by this method the snare is accurately applied over the tumor mass, then by gentle movements to and fro, or from side to side, depending upon the position of the tumor, it is brought as close to the point of attachment as possible. The wire is now carefully drawn taut and gentle traction is made to determine whether the tumor is held well in the grasp of the wire. If the tumor does not slip out of the loop, one is assured that the position of the instrument is correct. The next step in the procedure consists in removing the growth, and this is best accomplished by making gentle traction at the same time that the wire is drawn through. By this method the tumor is removed by a combination of cutting and pulling.

The entire operation can be performed in a few seconds; much shorter time, in fact, than is required to describe it.

For the removal of large tumors of the larynx, the usual method of using the snare should be modified. It becomes difficult and at times impossible to introduce a wire snare in such a manner that it will completely surround the entire mass, especially if but little space remains unoccupied by the growth. This difficulty may be satisfactorily overcome by proceeding in the following manner: Through the loop of wire one passes first a pair of laryngeal forceps, the handle of which may be locked. An assistant holds steadily the snare while the operator, passing the forceps into the larynx, grasps the tumor and locks them. The forceps are now passed to an assistant, the operator exchanging them for the snare. The tumor being held firmly by the forceps, it becomes a simple matter to pass the snare loop completely over the growth. This step may be facilitated by instructing the assistant to make gentle traction, thus drawing the tumor into the loop at the same time that the operator passes the loop around the tumor. (Figures 12, 13.) Having completely surrounded the neoplasm in this manner, the rest of the procedure is accomplished in the same manner that other snare operations are performed, as described above. In a large pedunculated carcinoma, situated in the opening of the larynx and attached to the right aryepiglottic fold and ventricular band, the author succeeded with great ease in removing the tumor en masse. Previous attempts at surrounding the tumor with the wire in the ordinary method had failed, owing to its size and position. (Figure 14.) This case was of unusual interest, inasmuch as its diagnosis was uncertain, even after pathologic examination. After removal there still remained an unexplainable infiltration, which was inconsistent with benign neoplasms. Subsequent developments proved the malignant nature of the growth.

Pedunculated carcinomata of the larynx are extremely infrequent. Two other cases have been reported, one by Ballenger and the other by Fraenkel.

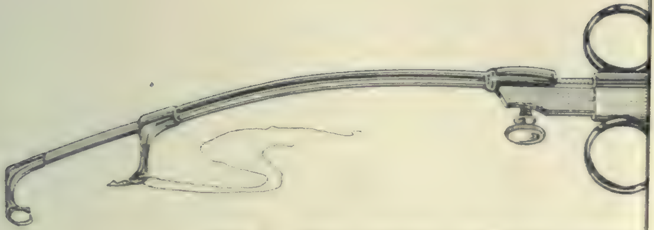


Fig. 1.—Pfau-Horsford epiglottis suture

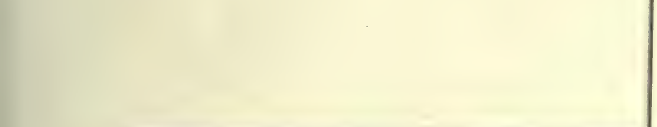


Fig. 2.—Chappell's syringe.

Fig. 4.—Caustic applicator.

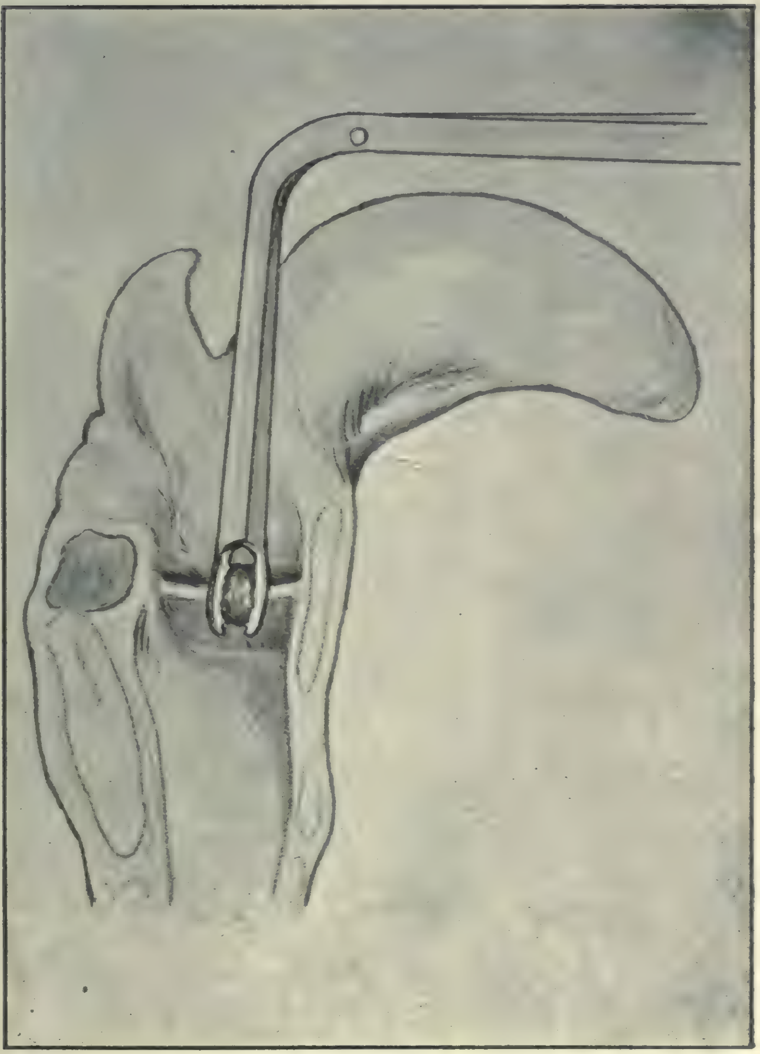


Fig. 8.—Showing removal of movable fibroangioma. Evulsion with Mackenzie forceps.

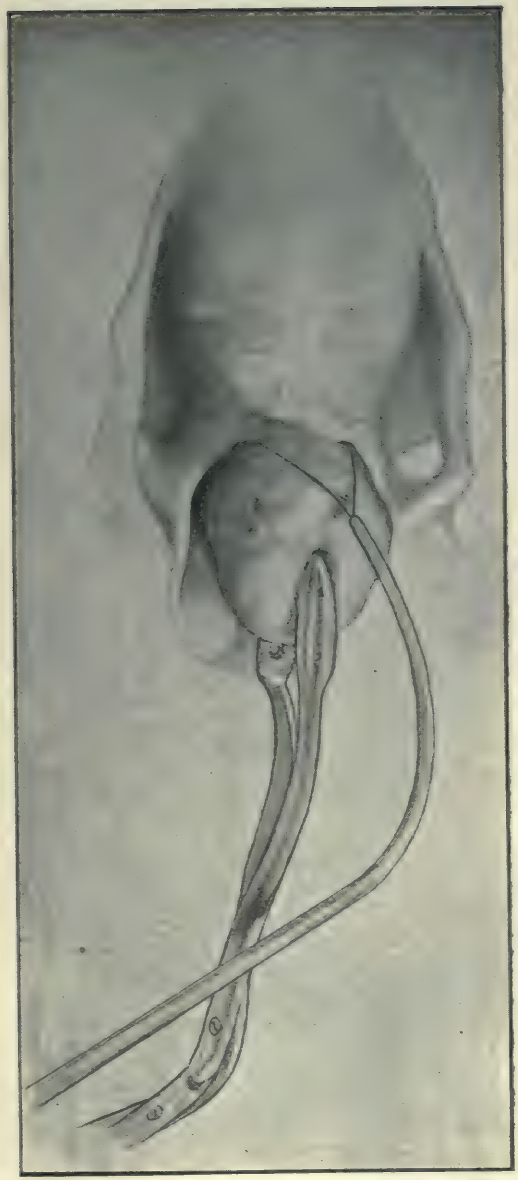


Fig. 13.—Removal of pedunculated laryngeal tumor. Showing snare and forceps in position.

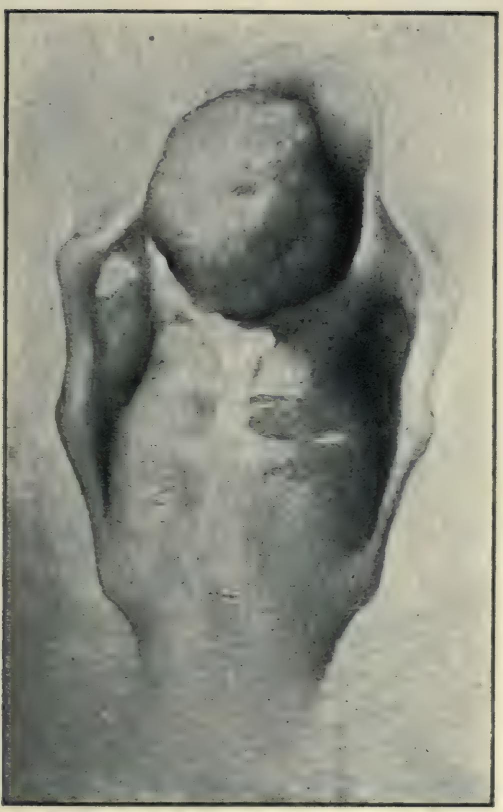


Fig. 12.—Large pedunculated carcinoma of the larynx.

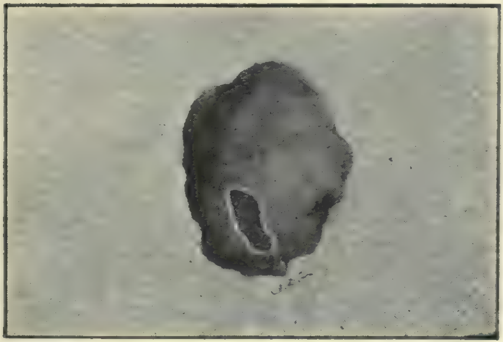


Fig. 14.—Pedunculated laryngeal tumor, life size. Some sections show an overgrowth of epithelium not sufficiently atypical to justify a diagnosis of malignancy. Other sections show small nests containing two and three epithelial cells which led to the diagnosis of carcinoma. The subsequent clinical history confirmed this diasgnosis.

V.

ACUTE SUPPURATION OF THE MIDDLE EAR—ITS NEGLECT AND PROPER TREATMENT.*

By Prof. Dr. Gustav Alexander,

Vienna.

Otitis media may be caused by a great number of different microorganisms. In the cases which have been published, every possible bacterial exciting cause has been found, sometimes in pure culture and sometimes as mixed infections. Saprophytes, also, have occasionally been found in the discharge, but in such cases we seem to have a secondary infection complicating the primary cause of the suppuration.

PATHOLOGIC ANATOMY.

In all cases where the middle ear is infected through the tube, we observe, first, a swelling of the tubal membrane which soon leads to obstruction of the tube. This swelling soon spreads to the mucous membrane of the middle ear and the tympanic cavity, as well as to the entire middle ear, viz., the meso- and hypotympanum, the attic, the antrum, and in many cases the pneumatic cells of the mastoid process become filled with an infectious viscid or hemorrhagic exudate. After a short time, sometimes a few hours only, suppuration is present, and the purulent inflammation of the mucous membrane of the tympanic cavity and drum leads to extensive exudation in the middle ear cavity. The pus finally breaks through the tympanic membrane and drains outward. During the earlier stages of the perforation, the secretion is chiefly of a bloody color, and not until later does it become clear, especially in influenza and typhoid otitis.

If the process runs a normal course, in about one week the secretion becomes a mixture of pus and mucus; the pus becomes ropy and more and more replaced by mucus, owing to the daily decrease of the secretion. The swelling of the mucous membrane of the tympanum and tympanic cavity decreases. The hole in the drum, in normal cases, closes after the secretion has ceased and complete restitutio ad integrum results, both anatomically and functionally. Sometimes in children, after a few weeks, the macerated epidermal crust is thrown off, leaving a fresh, normal, smooth and shining epidermal layer which has formed under it.

---

*Read by invitation before the British Medical Association, at the eighteenth annual meeting, Liverpool, 1912.

SYMPTOMS.

The prominent symptom, especially at the beginning of the disease, is the sudden, spontaneous and severe pain in the ear. This pain is often excruciating, and the patient is restless and sleepless. Sometimes, this severe pain is preceded by a sense of fullness and obstruction in the ear, as well as unilateral headache.

The loss of hearing reaches a high degree (speech, 1-2 m.) in the typical genuine otitis media suppurativa. Towards the end of the disease the hearing returns, and in uncomplicated cases becomes normal after the disease is cured.

At the beginning of the otitis media suppurativa, we usually find considerable fever, sometimes 40 degrees C. and higher. When the discharge of pus takes place, after the perforation of the tympanum, the temperature falls in cases running a normal course. An unfavorable symptom is a sudden, spontaneous fall of the temperature to normal or subnormal in the early stages of the inflammation, unless accompanied by a subsidence of all other symptoms of the disease. This is observed in cases of early intracranial affection, and is sometimes found in individuals lacking power of resistance or weakened by severe and general suffering from otitis media suppurativa. In early childhood, uncomplicated otitis media suppurativa often begins with vomiting and chills.

The condition of the tympanic membrane.—The otoscopic findings are a more or less pronounced swelling and redness of the tympanic membrane, which may involve the entire drum. In some cases, although the entire membrane may be inflamed, certain parts are involved to a greater extent, i. e., deep red color, severe swelling and bulging. Often extensive formation of vessels is observed. When the epitympanum alone is involved (Kümmell), the inflammatory symptoms are limited to the upper part of the tympanic membrane, i. e., to Schrapnell's membrane and the upper posterior quadrant, and often lead to bulging of the upper posterior part of the drum membrane (Politzer).

The deep red color of the tympanic membrane is by no means a reliable indication of the degree of the otitis media. Sometimes in a simple otitis media, and even in a myringitis, the tympanic membrane is of a deep red color, and greatly bulging. Owing to the bulging, the line of the handle of the hammer is entirely invisible. In cases of otitis media due to influenza, especially, the membrane may be of a deep red color even in very mild cases, and this may be accompanied by an elevation of the epidermis in the form of vesicles. On the other hand, the tympanic membrane may show a pale red color, even with a large collection of pus in the middle ear (empyema), and under certain circumstances may show only a radiate and peripheric vascular injection. In general, the intensity of the redness and bulging of the tympanic membrane decrease slightly with the progress of the purulent softening of the exudate in the middle ear. If the acute otitis media has led to an empyema of the middle ear cavities, especially of the tympanic cavity, without a perforation, the membrane sometimes appears opaque and grayish yellow, and will show only a slight injection of the radiate vessels of the tympanic membrane.

The expected perforation is generally indicated by a circumscribed yellow discoloration and marked bulging of the tympanic membrane. If it has taken place, we see an opening which is more or less round, allowing a free passage to the pus; or it may be a fissure, the edges of which approximate, just allowing the pus to escape. If the secretion is mopped out, it will sometimes be impossible to see the site of the perforation. We usually recognize it by the oozing of the secretion, though it may be ascertained by the use of Siegle's speculum. Sometimes the perforation is situated at the top of the mastoid process.

The size of the perforation varies. In the fulminating type of otitis media suppurativa accompanying acute infectious diseases, especially scarlet fever, we find comparatively large openings and in rare cases extensive destructions.

In advanced stages of suppuration the mucous membrane of the tympanic cavity resembles the tympanic membrane in redness, swelling and moisture. Later, that of the tympanic membrane decreases, and the epidermal layer becomes dry. Now, the perforation in the membrane is distinctly visible, even if such was not the case previously. The margin of the perforation is thicker than the surrounding tympanic membrane, but finally becomes of the same thickness. As soon as the perforation heals, the margin is grayish white.

The test of the function of the ear shows in every case a considerable decrease in sound conduction. At the beginning of the inflammation the sense of hearing is greatly diminished. At this time it is even possible to elicit a slight, spontaneous nystagmus to either side when looking in that direction, evidently in consequence of the pressure due to the secretion. It disappears with the appearance of the perforation.

DIAGNOSIS.

Even in the early stages of the disease the diagnosis of otitis media suppurativa can be made without any special difficulty, in most cases, from the anamnesis and the characteristic symptoms, viz., sudden appearance of violent pains in the ear, difficulty in hearing, fever, and the condition of the tympanic membrane.

From the standpoint of differential diagnosis, only the simple otitis media comes into consideration. Great bulging of the tympanic membrane, teatlike bulging of the upper posterior quadrant, especially circumscribed yellowish discoloration, and great loss of hearing, provided the hearing was normal previously; these symptoms speak against a simple otitis media and in favor of an otitis media suppurativa, as does the continuous pain, which at times becomes very severe. Attacks of pain with intervals free from pain speak more for a simple otitis media. High fever indicates otitis media suppurativa. In children up to the age of eight years, and especially in babies, we are unable to test the sense of hearing objectively and exactly. We may, however, make a test by means of instruments making a loud noise, or by means of sounds familiar to children, e. g., a bell, jangling of keys, sounding glasses and high pitched tuning forks, and thus ascertain approximately the degree of diminution of hearing. In some cases direct examinaton of the antrum (transillumination, X-ray examination) is of great service.

Little children complain of severe pain which they refer to the affected ear, but which is caused by late dentition, acute inflammation of the pharyngeal lymphatic glands, tonsillitis or the swelling of the pharyngeal tonsils. In some cases intense pains in the ear are an early symptom of an acute infection, usually following an affection of the mucous membrane of the oropharynx. At the time of puberty, violent pains in the ear sometimes indicate the beginning of an otosclerosis, if the otoscopic findings are negative.

In the stage of suppuration the odorless, profuse, pulsating and purulent secretion, and later on the odorless mucopus, speak for otitis media purulenta as opposed to otitis externa. If at the same time we find furunculosis of the external auditory meatus (otitis externa furunculosa), we may find it difficult at the first examination to make a diagnosis of whether we have to deal with a simple inflammation of the meatus, or with one complicated by an otitis media suppurativa, especially if we find multiple furunculosis of the external auditory canal with considerable purulent secretion.

In such cases the differential diagnosis is made on the basis of the following points:

(1) There is no fever in otitis externa of older children and adults; in small children we sometimes observe a moderate rise in temperature. If, therefore, in doubtful cases the anamnesis shows that the ear disease started with a high fever, we may assume, also, the existence of an otitis media.

(2) If in a case of otitis externa we cleanse the meatus and make a passageway by inserting a thin ear speculum, we will obtain a normal or slightly diminished aural acuity, if the drum is not too thickly covered with epidermis. If, however, there also exists an otitis media suppurativa, the hearing will not be improved after cleansing the external auditory canal.

(3) In children of four years or older, the bony auditory canal gives a good indication. The otitis externa always is confined to the membranous auditory canal. We find, therefore, after we have inserted an ear speculum through the narrowed part of the canal, that the bony auditory canal is of normal width and the tympanic membrane is unaltered. If,

on the other hand, we are dealing with an otitis media and a stenosis of the auditory canal caused by an affection of the antrum and the mastoid process, inspection of the bony auditory canal will show a sinking of the posterior upper wall, and the swelling of the external auditory canal is more pronounced in the bony part than in the membranous.

COURSE.

We distinguish three stages of the disease:

(1) The initial stage. This begins with the appearance of the ear symptoms and ends shortly afterwards, sometimes after a few hours, at the longest after three to four days, with perforation of the drum and purulent discharge in the external auditory canal. The violent pain and the fever usually last until the drum perforates and evacuation of the pus externally takes place.

(2) The second stage is characterized by suppuration. The patient is free from pain and the temperature is normal or slightly elevated. During the first few days there is a profuse purulent secretion. After about eight days the daily quantity of pus decreases spontaneously, and later on ceases. The secretion lasts for two to three weeks. During the last few days the secretion consists of almost pure mucus. The thicker and creamier the pus is and the more mucus the secretion contains and the less intense the secretion, the greater will be the danger that the secreted material will remain in the tympanic cavity or in the depths of the auditory canal, where it will either dry up or undergo putrefaction. The hearing will improve with the decrease of the suppuration.

(3) In the final stage, after the secretion has ceased, that is, one to two weeks later, the perforation will gradually close. Larger perforations require a longer time for healing; yet even with comparatively large perforations there is no danger of an opening remaining in the tympanic membrane, provided that the disease ran a normal course.

In all cases of otitis media suppurativa in individuals whose ears were normal up to the time of the disease, and where the disease appeared during the course of ordinary diseases, on the basis of chronic catarrhal processes in the nasopharynx, the prognosis is good for complete restoration of the ear, both anatomically and physiologically, provided the patient was treated correctly and did not suffer from any intercurrent disease.

The cases of otitis media suppurativa on the basis of chronic catarrhal processes in the middle ear have a tendency to recover with permanent changes on the tympanic membrane and diminution of hearing.

The same is true of cases of otitis media suppurativa due to adenoid vegetations in the nasopharynx and catarrhal changes in the eustachian tube, but they will last longer.

Finally, we must deal with the danger of persistent changes in the middle ear due to an organization of the mucous secretion during the final stage of suppuration. These are the cases where the otitis media suppurativa gives rise to a chronic adhesive process. There is the danger of permanent changes on the tympanic membrane in all cases which require repeated paracenteses, as well as in all cases in which there is ulceration of the tympanic membrane at the perforation, due to the poor state of nutrition of the patient (cachexia, anemia, chlorosis, etc.). Finally, there is the danger of persistent changes on the tympanic membrane in cases of recurrent otitis media suppurativa, which is often observed as a special type of otitis during infancy. The earlier attacks will recover with complete restoration of the tympanic membrane, but the later relapses will lead to a cicatrization; and after the sixth to the eighth relapse, healing will take place with a persistent perforation.

This is due to the fact that during the course of the disease the lamina propria of the tympanic membrane will gradually become atrophic, especially in the region of the perforation.

We must not forget that in any severe case of otitis media the purulent inflammatory stage may be followed by a catarrhal purely inflammatory stage.

All cases of otitis media suppurativa acuta which appear during the course of acute infectious diseases, have a tendency to become chronic.

This is especially true of the otitis of scarlet fever and of measles during infancy, as well as of otitis media suppurativa appearing during the course of diphtheria and of typhoid. Tubercular otitis media suppurativa, also, shows a striking tendency to become chronic, as do the otitis media suppurativa of the early stages of lues, and all cases of the disease appearing in otherwise weak, dyscratic or marasmic patients. A certain, though not great, tendency towards chronicity is to be found in cases of otitis media suppurativa of early infancy as well as of old age.

COMPLICATIONS.

In discussing the normal course of acute otitis media suppurativa, we saw that the inflammation, after the second week, is limited to the tympanic cavity and the antrum. We saw, also, that in normal cases the disease will involve the mastoid process and the eustachian tube only during its earlier stages. Complications are due to the fact that the mastoid process and the tube will take part in the inflammation during the later stages also, or that it extends beyond the limits of the anatomic middle ear and involves its vicinity. We distinguish therefore

(a) Intraaural complications.
(b) Extracranial complications.
(c) Endocranial complications.

Mastoiditis suppurativa is to be classed among the intraaural complications. Influenza otitis and typhoid otitis have a certain predisposition towards mastoiditis. Otitis caused by the streptomucosus, especially otitis caused by mucosus infection, is characterized by the rapid formation of an exudate which fills the entire middle ear, including the cells of the mastoid. Rapid softening of the bone is a characteristic feature of typhoid and influenza otitis. Mastoid processes with meningeal structure have a special tendency towards mastoiditis suppurativa. This leads to mastoid empyema through direct spreading of the pus. Diploetic mastoid processes are not so often attacked by the disease. In them, mastoid suppuration usually appears by metastasis. Rapid destruction of the bone is favored by rachitis during infancy, preceding constitutional diseases, as well as tuberculosis and lues. In tuberculosis, mastoiditis usually appears late and often has its origin in a tubercular periositis of the mastoid cortex. Retention of secretion in the tympanic cavity also favors the development of mastoiditis suppurativa from acute otitis media suppurativa. There is especially danger of mastoiditis in those cases in which the upper part of the tympanic cavity is primarily affected, and in which a small perforation in Schrapnell's membrane provides the only drainage, also in cases of mesotympanic otitis, in which the perforation is to be found in the pars tensa of the tympanic membrane, but not in the level of the membrane, but rather in the mastoid protuberance.

Attention is called here to the fact that in the latter cases, the mastoid proturbance should be destroyed by paracentesis.

Pyorrhea of the tube is to be considered as a complication of acute otitis media. It occurs in cases of tubercular otitis media suppurativa, as well as in cases of simple otitis in which the patients have large adenoids.

There is great danger of affection of the middle ear and of labyrinthitis suppurativa in cases of fulminating otitis media suppurativa which appear during the course of scarlet fever, measles, diphtheria and typhoid. Metastatic infectious neurolabyrinthitis may be caused by mumps or influenza.

Cases of hemorrhagic labyrinthitis are sometimes to be found in cases of otitis media suppurativa with peracute leukemia. The temporal relation between the otitis and the labyrinthitis varies greatly. If the two appear simultaneously, we have to deal with a panotitis. Usually, however, the purulent labyrinthitis is preceded by the otitis media suppurativa. In rare cases of meningitis cerebrospinalis, the labyrinth suppuration may be followed by the otitis. In the acute stages of tubercular otitis media, a labyrinth suppuration is not to be found.

Among the extracranial complications, special attention must be called to the burrowing abscesses. These cases also depend largely on the character of the infection, for in a comparatively large number of extracranial complications and burrowing abscesses, the streptococcus mucosus can be found.

The abscesses appear more especially in cases of pneumatic mastoid processes, in which the corticalis of the pneumatic mastoid is thin. In infancy, the formation of subperiosteal abscesses takes place with a striking rapidity. Their development is especially favored by rickets and scrofulosis in the patient. Sinking abscesses burrowing towards the external surface of the base of the skull are found especially in tubercular patients.

Among the endocranial complications of acute otitis media, we must give first place, on account of their frequency, to pachymeningitis and extradural abscess. They are found more frequently in the posterior than in the middle cranial fossa. If the extradural abscess is in the region of the sinus, we call it a perisinus abscess.

In the second place, we must mention sinus phlebitis, sinus thrombosis, pyemia, toxemia, and bacteremia.

About 50 per cent of all cases of inflammatory sinus phlebitis may be traced etiologically to an acute otitis media suppurativa. In the first year of life, however, sinus phlebitis complicating otitis media is rather rare. It is quite frequently found between the ages of ten and twenty, when it decreases in frequency but increases in danger as a complication.

In the first years of life we rather frequently meet with pachyleptomeningitis suppurativa in acute otitis media. In adults it is not so frequently a complication. Three types must be noted:

(1) Meningitis purulenta cum otitide media acuta, in which the meninges are affected simultaneously with the middle ear.

(2) Meningitis tuberculosa, of which there is always the danger in tubercular acute otitis media suppurativa.

(3) Meningitis secondary to acute otitis media suppurativa in older people, where the meningitis suppurativa appears acutely after an apparently favorable course of the acute otitis media. It usually ends fatally, sometimes after the primary disease has apparently gotten well.

In general we may say that there is greater danger of a meningitis in otitis media of the epitympanal type. In these cases the cavum epitympanicum is the primarily or exclusively affected part of the ear. The drainage of epitympanal otitis media is always difficult, and we are confronted with the special danger of a retention of secretion in the middle ear which greatly favors the extension of the purulent process to the meninges. In such a case, the disease usually extends into the middle cranial fossa, and in this connection the anatomic construction of the tegmen tympanicum is of importance. If it is either thin or dehiscent, the purulent process may make its way more directly and rapidly from the middle ear to the meninges than if the upper tympanic cavity were shut off from the dura by a compact bony covering. In cases of thin or dehiscent tegmen tympanicum, we will sometimes find intrameningeal abscesses of the middle cranial fossa during the course of the acute otitis media suppurativa. Purulent brain abscesses are very rare in acute otitis media suppurativa. Most cases of this kind in the literature will not stand criticism. Usually, it is a case of chronic otitis media suppurativa with an acute exacerbation.

If the acute otitis media is not treated, there is the danger of pus retention with its consequences. The stagnant pus becomes putrid and fetid. There is eczema of the auditory canal and inflammatory swelling of its lining, with granulations at the perforation, which may become occluded. The pus in the tympanic cavity is forced into the mastoid process. There is no doubt that some cases of purulent mastoiditis are caused mechanically by the stagnation of pus in the cavities of the middle ear. Moreover, the pus may exert considerable pressure on the walls of the tympanic cavity. The head feels heavy, there is pain in the temporal region, the tegmen tympani becomes inflamed, and finally extradural abscess and meningitis occur.

In other cases, the pressure upon the lateral wall of the labyrinth becomes very great, and we observe spontaneous nystagmus and vertigo.

In neglected cases of otitis media suppurativa, the hearing will always be greatly diminished, and the danger increases with the duration of the suppuration, since the hearing will remain diminished after the disease gets well. If a neglected case recovers spontaneously—and we cannot deny the possibility of spontaneous recovery of an acute otitis media—the stage of suppuration will frequently be followed by a long catarrhal stage. In consequence of the organization of the exudate, the otitis media suppurativa may be followed by the formation of bands of connective tissue in the middle ear, i. e., a chronic adhesive process, which in turn results in a degeneration of the organ of Corti and labyrinthine deafness. It may be briefly stated that complications of acute otitis media suppurativa are increased by neglect of the disease.

It has already been stated that a permanent pyorrhea may follow an incorrectly treated tubal suppuration.

TREATMENT.

In the first stage, we content ourselves with purely symptomatic local treatment.

Violent earache is sometimes considerably diminished by instillations of a 3 to 5 per cent carbolic glycerin solution or an adrenalin solution. If the auditory canal is wide, so there is no fear of a stenosis from epithelial maceration, we may instil a warm (40° C.) solution of aluminium acetate. Instillations of solutions of cocain, novocain and alypin have a good, though only temporary, effect. We use a tampon dipped in a 20 per cent solution, which is inserted as far as the tympanic membrane. The local application of cold (ice bag, etc.) is recommended during the first stage of the inflammation, while hemagogues are usually of no help. Rest in bed and a good evacuation of the bowels are ordered. Alcohol, strong coffee or tea are forbidden.

If the pain is very severe and is accompanied by fever, it will soon be seen that all anodynes are of no avail. In such cases it is advisable not to wait for spontaneous perforation, but to make an immediate paracentesis. A lance pointed knife mounted in a Politzer handle is used. The tympanic membrane is incised from below upwards, making the opening back of the umbo, and as long as possible.

If, on the other hand, circumscribed yellowish discoloration in the tympanic membrane indicates the imminence of spontaneous perforation, it will be necessary to make the paracentesis at that place, or at least to include it in the incision, otherwise it will be a useless paracentesis.

Sometimes it may be difficult to decide whether to make the incision or to wait.

If conservative treatment fails to give relief, i. e., if the pain and fever continues, paracentesis is indicated. As to the time to perform it, we must remember not to hesitate. It is better in doubtful cases to perform it than not. There is no danger so long as the operator observes all the aseptic precautions possible in the ear. The course of an uncomplicated inflammation of the middle ear may even be shortened by the fact that the serosanguinous exudate is evacuated externally through the opening, making its absorption unnecessary. Only in cases where aseptic precautions were not observed, do we meet with the danger that the uncomplicated inflammation may be converted into an infected purulent one through infection caused by the paracentesis. On the other hand, the omission of the paracentesis at the proper time, may cause an irretrievable injury, complications dangerous to life, and even death.

Preparations for performing the paracentesis.—The auricle is cleansed with a tampon dipped in benzene, and the head is then covered with a fenestrated sterile compress, through the opening of which the auricle is drawn. The external auditory meatus is washed with a 1 per cent solution of hydrogen peroxid. For anesthetizing, instillations of novocain-adrenalin solution is recommended. This is prepared by heating 1 cc. of a 20 per cent solution of novocain or alypin to 40° C. and adding shortly before using it five drops of an ordinary solution of adrenalin, heated. The solution should be left in the meatus ten to fifteen minutes. The ear clinic of Freiburg recommends the infiltration anesthesia of the drum by injecting a Schleich-adrenalin mixture into the tympanic cavity (Tiefenthal, Bernd).

Instillations of 5 to 10 per cent aqueous solutions of cocain, with an addition of five drops of adrenalin 1-1000, have also been recommended. Eucain (8 per cent aqueous solution) is innocuous and can be sterilized. Gray's solution is also worth mentioning (cocain. muriat. 0.5, ol. anilini, alcohol. absol., aa 5.0). Ten drops heated to 40° C. are left in the auditory canal about five minutes. Haug's solution is useful (cocain. muriat. 1.5 to 3.0, glycerin 10.0, alcohol. absol. 10.0).

The operation should be performed under aseptic precautions, since otherwise there is the danger of causing a secondary infection. If the paracentesis has been performed at the proper time, immediately afterwards there is an evacuation of a serosanguinous or sanguinopurulent exudate through the opening. If, on the other hand, pure pus is evacuated, it is a sign that the paracentesis was performed too late, that the inflammatory exudate has been subject to purulent degeneration to such a degree that all the cavities of the middle ear are filled with pus (acute empyema of the middle ear). Before performing the paracentesis, strips of gauze should be dipped into a warm solution of aluminium acetate, and these should be introduced into the meatus immediately after the operation, without any previous cleansing of the canal. A moist dressing is applied to the ear, and the patient should be put to bed. In cases running a favorable course, a profuse discharge of pus will be seen for the next few days. Within three to five days, the temperature will drop to normal, and there will be no pain a few hours after the operation.

If the perforation is not within the level of the tympanic membrane, but rather at the top of a mastoid protuberance, there will sometimes be an agglutination of the opening and symptoms of retention, in consequence of purulent stagnation in the mastoid process. In such cases the mastoid protuberance should be well incised with the paracentesis needle.

With the purulent secretion the otitis media suppurativa has entered into the second stage, and it is now necessary to take care of the discharge of pus and to prevent any retention or secretory stagnation. This is accomplished in different ways. Good success is obtained by moist dressings of acetic acid or 2 per cent alsol, renewed every day during the first few days. A moist strip of gauze is inserted into the external auditory meatus for the purpose of drainage, and after twenty-four hours we usually find the entire dressing saturated with pus. Later on, gauze strips 4 to 6 cm. long and 2 cm. wide are introduced. We may apply simple absorbent gauze dressings, or gauze impregnated with antiseptics (xeroform, vioform, dermatol, aristol, or ectogenous gauze). The insertion and removal of these gauze strips may be made by the nurse, the instrument, a blunt straight or angular forceps, being first sterilized by washing in benzene or by boiling. The dressing must be changed hourly or every other hour in case of profuse discharge, or two or three times daily if the discharge is slight. The point is that the strips should remain in the ear only so long as they absorb the secretion. If it is entirely saturated with pus, it must be removed before fresh pus collects behind it. We recognize that the dressing is being changed sufficiently often whenever there is little or no pus left in the external auditory meatus when the strip of gauze is removed, and whenever the patient is free from pain and the canal is open. Syringing is necessary only when the secretion is thick and ropy, and should be made with water at a temperature of 38° to 40° C. Insufflation of antiseptic powders is entirely unnecessary when the discharge is copious.

This method of treatment has the advantage that the physician is informed as to the condition of the suppuration, even if he doesn't see the patient daily. The latter will observe the decrease in the suppuration when the strips are not entirely saturated or are dry.

In a disease running a normal course, at the end of the first week of suppuration a viscid mucous secretion is found, which very soon thereafter becomes stringy. Therefore, simple removal of the gauze strips will be insufficient, since the semi-solid pus has the tendency to remain in the tympanic cavity or in the external auditory canal. It is now advisable to use hydrogen peroxid (3 to 6 per cent hydrogen peroxid, or perhydrol 0.5, aquæ destil. 30.0), which should be heated to 40° C. and dropped into the ear two or three times daily. The pus is removed from the tympanic cavity three or four times a week by politzeration, and subsequent cleansing of the external meatus is done with cotton pledgets moistened with a 5 per cent aqueous solution of perhydrol. The hearing is improved by the politzeration, though it will become temporarily worse when the cavities again become full of secretion. Cautious aspiration by means of a Siegle's speculum or a suction bell will sometimes be beneficial.

Special care must be taken in connection with the external auditory meatus, since an eczema or an inflammation of it would increase the purulent stagnation in the middle ear. In the later stages of the disease, the ear must be inflated daily, until the hearing is permanently good, and the lateral surface of the tympanic membrane is pale and dry.

When the suppuration has ceased, the perforation will usually close in a few days. The first sign of the healing of the perforation is that the edges become white. This color is due to the young epidermis.

The opening closes by gradual diminution. The growth of the new epidermis takes its course from the center of the tympanic membrane towards the periphery. All local treatment at this stage is contraindicated (Politzer). The closure of the perforation is spontaneous, and the tympanic membrane and hearing become normal. After an otitis media of longer duration, a scar or intermediate calcareous deposit may develop in the drum. It is advisable to keep the external auditory canal closed by a small plug of cotton for some weeks after the cure of the otitis media suppurativa.

In order to prevent repeated attacks of otitis media and also catarrhal relapses, it is necessary to treat the nasopharynx, to provide a free passageway through the posterior nares, to treat the epipharynx, and to restore normal conditions at the pharyngeal tubal orifices.

As to the treatment of otitis in nursing infants, it differs very little from that of later years. Early paracentesis is important, as otherwise we may be surprised by a sudden onset of cerebral symptoms (meningism), or even by a purulent meningitis. An important point is the very careful treatment of the external auditory meatus, the cutaneous covering of which must be greased repeatedly. Free drainage must be provided for by thorough removal of the secretion and the macerated epithelial substance, which collects in the form of small scales. If the canal is constricted or obstructed by eczematous swelling, inflammations, etc., the treatment of the otitis becomes more difficult, and there may be a retention of secretion with the most serious consequences. Repeated paracentesis is recommended in cases which are very prolonged. Granulations should be removed, for if allowed to remain the course of the disease is more prolonged, and it may even become chronic on account of proliferation of the granulation. After the patient has recovered, it is often necessary to remove the hypertrophied tonsillar or adenoid growths, in order to avoid repeated inflammations.

VI.

PNEUMOCOCCUS INFECTIONS OF THE NOSE AND THROAT.

By Clement F. Theisen, M. D.,

Albany.

During the past fall and the present winter, some rather unusual cases of pneumococcus infections of the nose and throat have occurred in the writer's practice. Some of the cases were similar in many of their clinical aspects to the cases occurring in the epidemics reported in Boston, New York and other places, last winter. The name "septic sore throat" was given to some of the cases reported last year.

Most of the cases observed by the writer ran the course of severe infectious conditions, with marked constitutional disturbances and involvement of the kidneys. A more or less severe kidney disturbance is not very uncommon in throat infections, such as peritonsillar abscess, follicular tonsillitis and other inflammatory conditions.

The following cases are characteristic of the type seen by the writer this winter:

Case 1.—Child, aged seven months, admitted to the Child's Hospital in December, 1912, for an apparently severe cold. The child had a rather severe cough and a profuse nasal discharge of a stringy character. No pus could be detected in the discharge. The temperature was 102° F. on admission to the hospital, and almost immediately great difficulty in breathing developed. On account of the character of the nasal discharge, cultures were taken from the nose and throat.

On examination of the throat, the entire pharynx, including the tonsillar region, soft palate and uvula, was found to be extremely edematous, and the mucous membrane was covered with the same stringy mucus. Because of the great edema, a satisfactory view of the larynx was not obtained. The glands on either side of the angle of the jaw were much swollen.

Two cultures from the throat were negative for Klebs-Loeffler bacilli, but contained pneumococci, and a swab taken from the nose alone, gave a pure culture of pneumococci. The difficulty in breathing for two nights was so extreme that we thought a tracheotomy would be necessary. There was undoubtedly a good deal of edema in the larynx also. That was six weeks ago, and there has been no recurrence of the difficult breathing. The edema of the pharynx gradually subsided under the use of a cold antiseptic spray. At no time was there any evidence of a collection of pus in the throat. The child is still in the hospital, and while the nasal discharge has stopped, there is still some fever. The blood count did not show any marked leucocytosis.

The child received one grain of urotropin in each ounce of fluid, for several weeks. Whether this modified the infection, I do not know. Reports as to the value of urotropin in air passage infections are contradictory, but it certainly seems to do good in some cases. The writer has never seen hematuria follow the administration of urotropin, although cases have been reported after only thirty or forty grains.

Case 2.—The next case is that of Mrs. H., aged 66 years, who said that she never had a sore throat until the one for which the writer was consulted developed. For years, from time to time, I have had occasion to treat her for chronic catarrhal otitis media. She has small submerged tonsils which had never given her trouble before. The attack started with a sharp chill and headache. There was severe dysphagia from the beginning of the attack.

Ordinary remedies were used for twenty-four hours, and when the writer was sent for the patient was sitting in a chair with labored stertorous breathing.

The throat presented almost the same appearance as in the other cases, i. e., great edema of the tonsils, soft palate, uvula; in fact, the throat looked like an inflated bladder.

Calomel was given at once, and after incision through the edematous parts, a culture was taken which showed a mixed pneumococcus and staphylococcus infection with the pneumococci greatly predominating. Albumin and granular and hyalin casts were found in the urine. Blood count showed a leucocytosis of 12,000. Ice packs were used around the neck, with ice in the mouth. An iced spray was used at short intervals, and urotropin was given, about forty grains daily being used, each tablet being dissolved in a large glass of water. Diet was limited to milk.

The edema of the pharynx persisted for a week, in spite of repeated incisions and local treatment. At no time was there any pus found. The throat gradually cleared up, but albumin persisted in the urine for some time. It is interesting that in this case the anterior surface of the epiglottis was also decidedly edematous, like the condition described by Michel[1] in 1878, to which he gave the name "angina epiglottidea anterior."

The writer in July, 1900,[2] also reported a series of cases in which the edema and inflammation of the anterior surface of the epiglottis occurred as a primary infection, without involvement of the pharynx.

In this series of cases, which also ran the course of severe acute infectious conditions, cultures showed mixed pneumococcus, staphylococcus and streptococcus infections, with the pneumococcus predominating.

Case 3.—The third case which the writer would like to report was similar to the last one, occurring in a man, aged 45 years, who had been subject to sore throat at times. This attack also started with a chill, headache and a sharp elevation of temperature (104° F.). The throat presented almost identically the same appearance as in the second case, except that the epiglottis was not involved. The great edema again persisted for a week, in spite of scarification and energetic local treatment. Albumin and casts were present in the urine, the spleen was enlarged, and in fact all the symptoms of a severe infection were present. Urotropin in large doses and in large quantities of water was given, and appeared to cut the attack short. Cultures in this case again showed almost a pure pneumococcus infection. The infection was so severe that Mulford's antipneumococcic serum was used, but without apparent effect. This case ran the same course as the second one, the throat slowly clearing up. Fairly high temperatures persisted for two weeks, and there was great prostration. No pus could be found in the throat at any time.

Cases 4 and 5.—Two cases, both young adults, were seen, with great inflammation and edema of the anterior surface of the epiglottis. The pharynx in both cases, with the exception of a slight redness, was normal. In both cases there was considerable difficulty in breathing, and great pain in swallowing; in fact, for several days they were practically unable to swallow. Cultures from both throats, after incisions, showed pneumococcus infections with a few other organisms.

These cases are of considerable interest, because they prove, contrary to Semon's[3] opinion, who claimed "that many of the forms of acute septic inflammation of the throat, that have been classified in the past as separate conditions, have a pathologic identity," that an acute edema of the anterior surface of the epiglottis may occur as a primary condition.

An interesting case of this kind has been reported by Crisp.[4] In this case, that of a boy eight years old, death occurred suddenly, apparently by suffocation, after he had been ill three or four days. The autopsy revealed a swelling of the epiglottis with no lesion in any other part.

Another interesting case has been reported by Fredet.[5] In this case, that of a young man of 20 years, great difficulty in breathing came on suddenly after a debauch. Suffocation rapidly ensued, and at the autopsy great edema of the epiglottis was found. When this was incised a large amount of pus was evacuated.

The fact that in the two cases of this kind, and in the three other cases reported by the writer in his paper, in all of which there was great edema associated with the symptoms of an acute infectious condition, and that they were all practically pure pneumococcus infections, is of some interest.

It is known that the pneumococcus, when some of the lower animals are inoculated with it, will sometimes produce local edema.

Welch,[6] in his interesting article on the "Micrococcus Lanceolatus," has brought this out. He quotes Foa, who distinguished a special variety of this organism. His pneumococcus he called diplococcus lanceolatus of the edematogenic variety. This edematogenic variety of the pneumococcus often produces, after subcutaneous inoculation into the subcutaneous tissue of some of the lower animals, widely spreading or local edema. Welch has also obtained cultures which when injected subcutaneously produced uniformly extensive local subcutaneous edema.

Another point of interest in this series of cases, is the absence of suppuration. In none of the cases was pus found when incisions were made.

REFERENCES.

1. Michel: Centralblatt für medicinische Wissenschaften, No. 2, 1878.
2. Theisen: Albany Medical Annals, July, 1900.
3. Semon: British Medical Journal, April 27 and May 18, 1895.
4. Crisp: Transactions of the Pathological Society, London, vol. 17, p. 28, 1866.
5. Fredet: Epiglottide suppuree avec oedeme de la glotte. Mort par. asphyxie. Soc. de med. leg. de France, vol. 7, p. 91, 1882.
6. Welch: Johns Hopkins Hospital Bulletin, December, 1892, p. 135.

VII.

TWO CASES OF AIR EMBOLUS FOLLOWING EXPLORATORY PUNCTURE OF THE ANTRUM OF HIGHMORE.

By H. M. Bowen, M. D.,

Toronto.

Exploratory puncture of the antrum of Highmore is looked upon as a very simple and harmless procedure, and is now practiced very extensively; but that serious and even fatal results may ensue, is shown by the following cases:

Case 1, March 5, 1910.—Patient, female, applied at the dispensary for treatment, and after the preliminary examination it was decided to wash out the antrum of Highmore. An application of a 20 per cent solution of cocain was made to the inferior meati and about fifteen minutes given for cocainization.

The puncture was made with some difficulty, owing to the unusual thickness of the bone. After inserting the needle, it was found impossible to force the air through with the syringe, so the needle was withdrawn slightly for a second attempt, which was also unsuccessful. During the third attempt the patient, without the slightest warning, became rigid and cyanotic, resembling very much an attack of epilepsy. A moment or so later there were convulsive movements of the extremities, frothing at the mouth, and stertorous breathing.

She was placed in the recumbent position, with the expectation that consciousness would return in the course of a few moments, but, on the contrary, she remained in this condition for seventy-two hours, and did not become rational until the evening of the fifth day.

As she did not respond to the ordinary stimulants, she was admitted to the hospital for observation. At eleven o'clock that night the temperature was 101 2-5°, pulse 75, and respirations 20. The temperature remained around 100° until the third day. She vomited the first evening, and again the evening of the third day. There was profuse perspiration, especially at night, and muscular twitching almost continuously. Occasionally she became quite violent and required the use of a restraining sheet. During the fourth day there was involuntary micturition, and during the lucid period that evening she complained of pain in the abdomen. During the fourth day a partial paralysis of the left arm was apparent. She was able to sit up in bed on the fifth day and would converse when spoken to. She was discharged on the seventh day.

Case 2, July 25, 1911.—Patient, male, age 24 years, over medium height, muscular physique. His application for the police force of New York had not been considered, owing to defective nasal breathing.

Examination revealed a marked deviation of the septum for the relief of which he submitted to a submucous resection.

One month later he again applied at the dispensary, complaining of nasal discharge and alternating nasal obstruction.

The inferior turbinate on each side appeared largely swollen and of a dull blue color. The outer wall of the middle meatus had a peculiar dull gray, snail track-like appearance, presumably due to the flow of mucopus down the middle meatus, and under the assumption that one of the accessory sinuses was infected, it was decided to begin the search for the pus by puncturing the antrum.

Accordingly, the inferior meati were cocainized with a 20 per cent solution. The right side was washed out with a negative result. The left side was punctured in the same manner, but as the air was forced in with the syringe the patient became rigid, eyes half closed, eyeballs rolled backward, then relaxation, profound cyanosis, and slight muscular twitching. He was placed in the recumbent position, and outside of a few gasping respirations, at the rate of about three per minute, there was little evidence of life.

He was immediately given a hypodermic of strychnin, adrenalin, minims xv, several hypodermics of whiskey, artificial respiration being kept up all the while.

At the end of about twenty minutes the cyanosis cleared somewhat and it was thought that the danger was over, but in a few minutes it deepened again, and death occurred approximately one hour after the puncture.

A postmortem was performed the same evening. When the calvarium was removed, the sinuses of the dura were revealed, greatly distended, but there were no apparent changes around or in the substance of the medulla. Examination of the abdominal contents revealed old adhesions around the appendix and gall bladder.

When the sternum was removed, the pericardium immediately bulged forward into the opening. An incision into the pulmonary artery was followed by a sharp hissing sound of escaping air and a collapse of the distended heart.

The cause of death was quite apparent, but the condition of the antrum was most interesting of all. The puncture in the antrum wall was easily found, and when the anterior wall was removed, the mucous membrane of the roof, except for a small part, as well as the outer and inner walls, was found detached.

The mucous membrane covering the roof was hanging about a quarter of an inch below, and it showed a small puncture mark. The bone of the roof and posterior wall did not show any puncture mark, consequently the needle did not penetrate beyond the antrum.

The inner wall of the antrum was removed and the ethmoid was cleared out up as far as the sphenoid, but no large or misplaced vessels were found. There was not the slightest trace of hemorrhage in the antrum.

Taking everything into consideration, the only explanation which could account for the passage of air into the general circulation, would be that the needle penetrated the mucous membrane of the roof and the air was forced between the mucous membrane and the bone, opening up numerous capillaries.

Up until this time the technic employed was to blow air through the antrum before washing out. In this case there was no unusual force employed in blowing the air through, consequently it will readily be seen that it is a very dangerous practice. It does not indicate anything further or accomplish anything in preference to the solution.

In using the solution first, providing the needle did puncture the mucous membrane of the outer wall, no harm would be done by the injection of a small amount of sterile saline solution into the general circulation.

VIII.

NASAL HEMORRHAGE FOLLOWING TURBINECTOMY IN A HEMOPHILIAC TREATED BY THE INJECTION OF HUMAN BLOOD SERUM.

By Linn Emerson, M. D.,

Orange.

The use of human or other blood serums in cases of hemophilia is proving to be a measure of value. In the journal of the American Medical Association of October 26, 1912, four cases are reported by different authors, in which the measure was apparently successful in three.

Dr. Clement F. Theisen has also reported several cases. Dr. J. H. Bradshaw of Orange reported to the Orange Memorial Hospital Medical Society in September a case of hemophilia in the newborn, saved by the use of human serum, and described the apparatus used by Welch and himself to secure the blood for the serum.

Mr. J. C., aged 31 years, a vigorous, healthy married man, consulted me on September 28, 1912, for chronic catarrhal deafness. To improve hearing and facilitate the passage of the eustachian catheter, I removed the major portion of the left inferior turbinate on the morning of October 6th. As the bleeding was rather free and continued to ooze even after packing with Simpson tampons of Bernays' sponge, I advised him to remain in my office during the afternoon, as I was called out of town to perform several adenoid operations.

I had just begun my second adenoid operation when I received a call from his family physician, who was with him in my office, and who seemed to think the case needed my immediate attention. As I was more than twenty miles distant and had four other adenoid operations scheduled, all of whom had been starved and prepared, I could not see the feasibility of an immediate return, but advised him to either pack the patient himself or get some other rhinologist. This he was unable to do, it being late Sunday afternoon and no other specialist being available.

I suggested serum injection, and the patient was given 2000 units of diphtheria antitoxin. On my return at 8:20 p. m., this was repeated, and the bleeding being persistent, I removed the tampons and packed tightly with a cotton tampon behind and gauze saturated with adrenalin in front. Even after this packing the blood oozed steadily from the front, and one or two ounces were lost during the night.

A special nurse was secured and the patient remained in bed in my offices, and I also slept there that night. About noon the following day the hemorrhage being nearly stopped, he was taken to the Orange Memorial Hospital in an ambulance.

The slightest movement or lying down would cause the oozing to start afresh from the front, so the patient sat up in bed for more than a week after the operation.

On the fourth day after he was plugged, I removed the posterior plug without mishap, but he was so fearful that the removal of the gauze would cause renewed bleeding, that he was permitted to pull it out himself and cut it off an inch at a time. Ten days elapsed before the gauze all came away, and the day following he was allowed to go home. Despite the fact that he remained very quiet most of the time in bed, bleeding recurred on the 20th, two weeks from the day of the operation, and after three days of milder methods of treatment I was obliged to plug him tightly again at 3 a. m. on the 23d. On this same day the patient's brother was brought from a distance, and I called Dr. Bradshaw to get blood from him for the serum. The apparatus did not work satisfactorily, and most of the blood was collected by spontaneous flow from the median basilic vein.

The serum was separated by clotting and gravity, and about seven ounces were secured. The first two days 20 cc. were injected subcutaneously, three times a day, and the effect immediately following the injection was marked. After two days the frequency of the injection was lessened, and when reduced to once daily the bleeding recurred, but to a much lesser degree.

The patient's physical and mental condition were becoming alarming, so it was decided to secure more serum. A healthy man was secured, and on October 30th I bled him from the median basilic vein, collecting the blood in a wide-mouthed dressing jar; when the blood spurted, it was deflected into the jar by the cover held by a nurse. More than a quart of blood was secured, and about nine ounces of serum was obtained. This was used as before, and after using 20 cc. three times a day for three days, the packing was finally completely removed on November 2d. The injection was continued once daily for nearly a week after this.

The patient has very slowly and gradually regained his health and strength, but has been very apprehensive of a return of the hemorrhage. As late as February 1st he came to me, greatly worried because the mucus from his nostril was occasionally slightly tinged with blood.

His early history, secured by careful questioning after the operation, is interesting. He once had a tooth extracted and bled for three days afterward, but does not seem to have lost any very great amount of blood.

While a college student his leg was bruised while playing football, and a large swelling developed which "laid him up" for nearly a year. Several physicians were much puzzled at this condition, which was no doubt an intramuscular hemorrhage.

He had seven and one-half grains of calcium chlorid three times a day and gelatin dessert with his meals for a week before operation. This was also kept up for a number of weeks during his convalescence.

It is interesting to note that about a pint of serum, more than half of which came from a donor unrelated by blood, gave rise to no unfavorable symptoms of any sort.

IX.

ON THE FUNCTION OF THE TONSILS.*

By Louis M. Freedman, M. D.,

Boston.

In the consideration of the function of the tonsils, we are entering one of the medical fields of controversy which has lasted a long time. It has been difficult to ascribe a function to a tissue which, after its removal from the body, has not apparently been missed. The main cause of this controversy has been whether we should interpret tonsillar conditions in a pathologic, injurious light, or in a benignant, salutary light. There have been many theories regarding tonsillar function, none of which have in any way been definitely proven true. Yet, we are more definitely certain that the activity of the tonsils as such are of temporary use at least. We are at least certain enough about a possible function to make us hesitate in the use of radical tonsil surgery.

To better understand the possible functions, we must briefly consider, in outline at least, the anatomy of the tonsils. The tonsils are composed of lymphatic cells, collected in groups called follicles. These follicles are separated from one another by areas of connective tissue. These masses so formed are held together externally by a fibrous capsule which binds the tonsil to the tonsillar sinus—the triangular space between the anterior and posterior pillars of the soft palate. The internal surface of the tonsil, facing the oral cavity, is lined by a flat epithelium. The epithelium of the mouth and nasopharynx is generally ciliated, and directs particularly toward the tonsils. The surface of the tonsil is irregular and indented with crypts, ten to twenty in number, which have opening into them the ducts of the muciparous glands situated in the stroma of the tonsil. Surrounding each follicle is a close plexus of lymphatic vessels. From these plexuses the lymphatic vessels pass to the submaxillary lymph glands below the angle of the jaw. From the submaxillary lymph glands the lymph goes to the deep cervical glands. The main blood supply of the tonsil is the tonsillar artery, a branch of the facial artery, although many other vessels contribute in part to the arterial supply of the tonsil and its surrounding tissue.

---

*Read at the fall clinical meeting of the Staff of Mt. Sinai Hospital, Boston, November, 1912.

In development the tonsil is not quite mature at birth; but at the end of the first year it is fully formed and active. If it functions at all in any special way, it does so in the early years of life, most likely before the age of seven. After that the tendency is for it to atrophy. A tonsil remaining large after that period is generally not found to be normal, at least in its activity. Thus, in the tonsil we have a lymphoid structure with a short period of usefulness.

In the study of the physiology of the tonsil, many hypotheses have been advanced concerning its function; some of these are not rational, others are very suggestive. Flak holds that the tonsils are residual embryonic remains. In favor of this is urged the fact that in intrauterine life the tonsil becomes markedly developed, and in early life it tends to disappear. Against this is the fact that the tonsils have been traced in all the animal forms from reptile to man, and it is most highly developed in man. Another old theory was that the tonsils had some necessary and obscure relationship to the organs of generation, and removal of the tonsils would cause sterility. The latter part of this statement has been absolutely proved untrue. Whatever possible relationship there exists between these tissues, it is certainly not a necessary one.

The theory that the tonsil is a blood forming organ in early life, and that after the tonsil has passed its active period this function goes over to the bone marrow, has had many supporters. Harrison Allen holds that the tonsils are a tissue of the type we see in the thymus, lymph glands, and the bone marrow. When the thymus retrogrades, the tonsils develop and take up its function; these in turn retrograde and in turn the bone marrow takes up the function at the full growth. This is not rational; for, long before the thymus has reached such a stage, the tonsils and lymph glands are not only developed but hypertrophied. Further, to take this viewpoint would mean to consider the hypertrophied tonsil most active at a time when it should be retrograding; yet under such conditions it does not alter or lessen the activity of the bone marrow. Further, even if it were possible that the thymus, tonsils and bone marrow might be active vicariously at the unstated periods, it is still anatomically unreasonable; for, structurally they are each different. The thymus and bone marrow each lack germinal centers; the bone marrow lacks follicles; and in the lymph glands are lacking the various stages of development of the red blood corpuscles which we find in the thymus and the bone marrow. The anatomic differences lead to the conclusion that the tonsils and the lymph glands are the origin of the lymphocytes, and the bone marrow of the myelocytes. We have no evidence to show that the tonsils are of great significance in blood building. But the anatomic similarity between the tonsils and the lymph glands is so great, that one must admit that the tonsils are a part of the lymphatic structure and its system. Wood considers the tonsil a lymphocyte forming organ. According to him, the origin of the lymphocytes is not in the germinal centers, but rather the epithelium of the tonsillar crypts. He has seen all the transitional forms to leucocytes. In this belief he is apparently alone. The crypts are lined with a stratified epithelium which is not of mesoblastic origin. To agree with Wood is to overthrow all our views in relation to the mesoblastic origin of the leucocytes.

That the tonsil is a secreting organ many sought to prove. Some thought that it held a digestive ferment which converted starch to sugar. No secretion of such a nature has been obtained. Others thought that the tonsil secreted a mucus which facilitates deglutition by moistening the bolus of food. Likewise, this has never been demonstrated. Finally, an internal secretion such as the thyroid and adrenals have, has been sought for in the tonsils. Masini injected tonsillar extracts into animals, and thought he obtained a rise of blood pressure such as is seen with adrenal extracts. Scheier, in covering the same ground, got a notable fall in blood pressure. Pugnat, repeating these experiments, got neither rise nor fall in blood pressure. It is evident, therefore, that with the three possible conclusions obtained by three different men, that at least two of them probably had some error in technic that led them to the wrong result.

The late Dr. Nelson of the Harvard Medical School and myself undertook to make tonsillar extracts, for this purpose. We divided the work into two parts. First, we made extracts of the extracellular substances; second, we made extracts of the intracellular substances. The extracellular extract was made by chopping up the tonsils finely, and rubbing them up with sand mixed with .8 per cent normal saline in a mortar. This was allowed to stand for a few hours and then filtered. The filtrate was then injected into the jugular vein of a properly anesthetized rabbit, so placed that the recording apparatus for blood pressure and respiration would register the changes. The extract was injected in varying doses and at varying intervals, but no change of any sort was noted. By way of control, a small portion of the filtrate was treated with alcohol and a precipitate obtained, showing the probable presence of an albuminous substance, and that the filtrate was not absolutely inert. Further, as another control a 1-10,000 solution of adrenalin was injected into the rabbit in very small amount. This showed a tremendous rise in blood pressure. This showed quite definitely that the failure to get an effect upon the blood pressure with the extracellular extract was not due to the inertness of this extract, or to any difficulty with this particular animal, but rather because an aqueous or normal saline extract of the tonsil does not contain any blood pressure altering substances. The extract for intracellular substances was made by similarly cutting up the tonsillar tissue very finely; but this time, instead of the saline solution, a solution of cupric chlorid was used to dissolve away the cellular envelope. This was allowed to stand forty-eight hours and then filtered. The clear blue filtrate was then treated with H_2S gas to precipitate the sulphid and thus remove the copper salts from possible activity. The colorless filtrate resulting was then injected into a rabbit, under the same condition as before stated for the salt solution extract. The result was likewise negative. The same controls were used as with the salt solution extract, with the same result. In doing this work we found how easy it was to be led astray by a slight error in technic. For instance, the first extract we used with cupric chlorid, we did not free entirely of its copper content. This resulted in causing a marked fall of blood pressure in the first part of the curve, which no longer occurred when the rest of the filtrate was freed of the copper content entirely. It would seem then from the foregoing, that no internal secretion which affects blood pressure is present. This certainly agrees with our clinical experience. For, with tonsillectomy being so widely practiced in America, almost to the exclusion of any other method among some very active operators, we have as yet not been able to discover that any necessary secretion has been lost; or, at least, if such a secretion has been removed, other organs continue to supply it in sufficient amount for the individual, so that no loss is felt.

We come now to the theories that give to the tonsils a protective character for the body, in contradistinction to those theories which have for their basis that the tonsil is a portal of entry for infection, and therefore a source of danger.

Ashhurst holds that the tonsil is a protective organ, but from an eliminative point of view. To support his view, he finds it significant that in diphtheria, scarlet fever, and other exanthemata, the tonsils are affected after other manifestations of the disease have been present days or hours. To further support his view, he cites the case of a physician who pricked his finger when opening a postscarlatinal abscess. The next day he attended another septic case, during which he noticed that that finger was uncomfortable. Following that, his finger became swollen, and his arm likewise, with a resulting lymphangitis. Then constitutional symptoms such as slight fever and malaise intervened. Two days after that the tonsil of the same side became inflamed, and later the tonsil of the opposite side also. But no cultures were taken. This he considers a sort of elimination. In the same way he explains the obscure relation between articular rheumatism and tonsillitis, and endocarditis. The position does not seem tenable. In the diphtherias and the exanthems it has been as frequently noted that the tonsil affection precedes the other symptoms as it follows them. Anyone who has treated many of these cases has had for a few days or hours, no more than the throat affection and temperature to go by for a diagnosis. This is certainly true of the diphtherias. In measles, the coryza and lacrimation are the first signs. So likewise we more frequently see the tonsillitis anticipate the articular signs in rheumatism than follow them. Further, the case of the physician cited is open to a number of possible interpretations, of which the elimination theory is the least likely of all the solutions. With no culture taken there is not even the evidence definitely that the same organism was present in the arm as in the tonsils. Is it not more likely that the organisms already present in the tonsils received new stimulation for activity with the lessened general body resistance of the patient, when an active septic process was present in the arm? Ashhurst further holds that the tonsillar hypertrophy is not simply a local condition, but is rather evidence of a diathetic taint or condition of malnutrition. Removal of such hypertrophy would be a benefit to the general health by removing a mechanical obstruction to respiration; and secondly, it would improve the function of the tonsil, whose crypts and recesses are, in the process of hypertrophy, made deeper, and their normal outflow thereby impeded. The accumulations in these recesses are a source of danger to the entire organism. It is difficult to be contented with the supposition that tonsillar hypertrophy is evidence of a diathetic taint; for, if that be true, a tremendously large proportion of the children of America and Europe have that taint. Further, we see it as frequently in robust and well nourished children as in poorly developed types. It would seem that tonsillar hypertrophy might in fact be an evidence of beneficence, if we believe in the doctrine of the "survival of the fittest."

It has been a much mooted question whether the normal direction of the lymph flow in the tonsil is inward or outward; and on the answer to this question depends the protective character of the tonsil. We have seen that the Ashhurst view is that the protection is by a sort of elimination outward.

Stöhr discovered that through the epithelium of the tonsil there is a constant stream of leucocytes wandering forth. The leucocytes swarm about like patrols or watches, and fall upon the foreign particles, take them up and destroy them by phagocytosis. Brieger, however, showed that it was not the leucocytes which come forth, but rather the lymphocytes, and they are not supposed by the physiologists to be phagocytic in character. Further, he contends that the lymphocytes do not of themselves actively and independently change their position, but are passively transported from the tissue of the tonsils. This medium of transportation can be nothing other than the lymph fluid which fills the interstices even through the finest structure. This is not really emigration, according to him, but rather a streaming through, or "Durchstrüming," as he calls it. The protective quality is due, first, to the mechanical outflow of the lymph, which thereby hinders the entrance of foreign substances; and secondly, to the bactericidal effect of the lymph fluid, which is identical with the blood serum; thirdly, it is due to bactericidal substances set free in the decomposition of the lymphocytes after their exit from the tonsil. Stöhr does not agree with this. He says that the lymphocytes wander out through the epithelium, and the places where they come through may serve as entrance points for bacteria and foreign substances, and thus are a source of infection in the form of "physiologic wounds or spaces." From the Brieger viewpoint, the lymph fills out these spaces so that they are not a source of infection. It is to be admitted that the crypts are a great source for infection; for they are filled with collections of bacteria, detritus, and whatever foreign substances might enter from the throat. It is, however, difficult to believe with Stöhr, that any tissue of the body is formed primarily to be a source for infection.

Goodale experimented with the absorptive power of the tonsil for foreign particles. He injected into the crypts of hypertrophied tonsils, under moderate pressure, a watery solution of carmin in fine suspension, by means of a dull pointed cannula attached to a hypodermic syringe. The tonsils were removed at varying intervals following the injection. The first ones were removed twenty minutes after the injection, the next forty-five minutes after injection, the third an hour, etc., and the last ten days after the injection. The tonsils were imbedded and serial sections cut. The sections showed the carmin in the crypts, from which fine lines radiated up to the epithelium. Where the structure of the epithelium was loose, the particles entered deeper into the cellular layers. Some of the particles were close to the leucocytes, others were within the leucocytes. How deeply the carmin entered into the tissue, depended upon how long after the injection the tonsil was excised, and the looseness of the epithelial layers. In no case were the particles found within the follicles. In the case where the carmin had been injected ten days before, there was very little carmin in the crypts; but most of the carmin was in the deeper interfollicular tissue, radiating in the direction of the deeper connective tissue layers. Each case showed bacteria in the crypts, but none in the tonsillar structure. In one case a solution of carmin in which a characteristic bacillus had developed was injected. Two days later the tonsil was removed and examined. The bacillus was found in great numbers in the crypts, free and in neutrophiles; yet none were found in the tonsillar tissue, although in the interfollicular spaces much carmin was found. Further, four cases of acute follicular tonsillitis were examined histologically for bacteria. Though many bacteria were found in the crypts and mucous membrane surface, none were found in the tonsillar tissue. Brieger objected to these results, on the ground that possibly undue pressure had been used in forcing the fluid into the crypts, so that it was not absorption but pressure that brought the carmin into the tonsillar tissue. But in this he was wrong; for the first tonsil, removed after twenty minutes, showed only the presence of carmin in the crypts, not the tonsillar tissue. Further, had pressure been the actual means of introduction into the tonsillar tissue, then the bacteria would also have been unavoidably forced along with the coloring matter. These experiments, therefore, show that the tonsils absorb foreign particles, but not bacteria; and that inflammations in tonsils are primarily set up, not by the bacteria, but rather by their toxins.

Schoeneman considers the tonsil a lymph gland, like the rest in the body, only it is submucous in position and has for its main function the drainage of the nasal mucous membrane. This he proves by painting the nasal mucous membrane of one side with coloring matter, which he later finds in the tonsil of that side. He likewise explains the tonsillitis which follows the socalled head colds; and so also the tonsillitis which follows operative measures on the nose. Tonsillar hypertrophy is to him comparable to simple chronic lymphadenitis.

Further, on the absorbent power of the tonsil, George Wright of Boston has shown a definite relationship to the teeth. He shows that the enlargement of the tonsils without necessary inflammation may occur, and usually does, at the correspondingly same periods as the four periods of molar dentition: at two years, six years, twelve years, and at seventeen years. Prophylactic treatment of the teeth at these periods will cause reduction of the tonsillar enlargement. In the development of the teeth at these periods, a dental sac, analogous to the placenta, is formed to carry all the materials necessary for the production of the enamel and dentine and their nutrition. "The final disposition of what remains of this organ after the teeth have erupted, together with the bone and connective tissue surrounding, also the roots of the temporary teeth are involved in lymphatic absorption. With the upbuilding of forty-eight teeth and the changes occurring in the jaws, there is likewise much waste tissue for disposal." Wright believes that the tonsils together with the lymph glands take care of these waste substances. So likewise, with decayed teeth, the tonsils are enlarged to absorb the waste and septic material. If the teeth are cleaned up, thus leaving less for the tonsil to absorb, the tonsillar enlargement does not occur or subsides.

Jonathan Wright finds, from an evolutionary point of view, something significant in the enlargement of the tonsils in civilized children in response to changes of temperature from the overheated house to the excessive cold out of doors. In the system there is a call made upon the vasomotor and cellular activities; in the response to this we get an ontogenetic and phylogenetic adaptation. This response is usually a congestion, and often an inflammation, resulting in the hypertrophy of the pharyngeal lymphoid tissue. Enlarged tonsils, as has already been previously noted, are as much if not more often present in robust as in frail children. It is also a fact that the submerged tonsil is more the tonsil of infection than the large free one. The relatively greater frequency of enlarged tonsils among the children of civilized man, apart from the barbarous races, goes hand in hand with their greater natural immunity against many infectious processes. It is a well-known fact that the American Indian has not been prone to have large tonsils; and yet, the ordinary infectious diseases of childhood have a more serious effect upon him.

R. H. Good, and likewise Sheedy, think that the tonsil may in early life act so as to immunize the system against many diseases, the crypts acting like culture tubes in which various toxins are elaborated and introduced into the system in quantities sufficient for immunization. He carries it still further, by calling attention to the fact that children are more likely to have certain contagious diseases that adults rarely get—due to the immunity established thus in childhood. Because of the location of the tonsils, bacteria entering the oronasal cavity are directed toward the tonsillar crypts. The crypts are simply culture tubes or receptacles in which bacteria of every variety may be cultivated—the mucus derived from the glands outside of the tonsillar structure, acting as a culture medium. The bacteria multiply in the crypts, give off products (vaccines) which are taken into the tonsil by the lymph currents and thence into the entire system. When these products come in contact with the fixed tissue cells, antibodies, agglutinins, opsonins and so forth are produced. Thus are the leucocytes prepared to exert their phagocytic action. This process of leaving the bacteria in the crypts long enough to establish an immunity and then be carried off by the phagocytes produces a socalled tonsillar equilibrium.

With so many possible theories, a number of which bear the stamp of having at least a kernel of truth and plausibility, it is very difficult to properly come to any definite conclusion in regard to the functional properties of the tonsils. Yet some facts stand out quite definitely. It is evident that the tonsil is a lymphoid structure such as any lymph gland; it is connected with the lymph glands and channels, and has the advantage or disadvantage of being superficially placed. But it is apparent that its period of activity is only for the early years of life. It is very likely that the tonsils play some part in blood building, but that is not very important for the system, because it is not the only organ for this purpose, and also because it is apparently not the important part of its work. It is reasonable to believe, because of the leucocytes present and their noted phagocytic character, that they are protective during their period of normal activity. The experiments of Goodale showed quite conclusively that this protective character against bacteria was quite definite. It seems fair to call the tonsils, as many have, "the sentinels of the body." When an infection enters through the tonsils, there is great likelihood that the tonsil attempts to overcome the invaders, and failing in the attempt, the next lymph glands in order of defense—the submaxillary—take up the battle; and if any of the invaders sift through further, the cervical lymph glands become the defenders. Yet it must be noted that in the usual tonsil infection it is not the bacteria but their toxins which have apparently set up the inflammation. Whether a process of immunization actually takes place, of course, is not definitely proven, but there is certainly much in favor of such a theory. However, if the tonsil is so diseased that its ability to cope with an infection is so far reduced that on very slight provocation it becomes inflamed, and too frequently in the battle between it and the invading bacteria it becomes the loser, so that constitutional symptoms intervene, it is then fair to presume that no longer is it a sentinel, but rather an obstacle to defense, and may then actually serve as a portal of entry for disease.

The tonsil is so often spoken of as a portal of entry for disease that many have come to the conclusion that this is its function. That, on the surface, is not rational. It is hard to believe that the particular function of any organ or tissue of the body should essentially be injurious. It is, however. reasonable to believe that through disease any organ may be perverted in its function and thus serve as a source of danger to the entire system. Lexer painted virulent cultures of staphylococcus, pneumococcus and streptococcus on the tonsils, and set up an inflammation in the tonsil and resultant constitutional symptoms; he did the same on the mucous membrane of the throat, with a resultant inflammation and constitutional symptoms. This makes it quite evident that the tonsils per se are not simply portals of infection; for, given the same conditions, the pharyngeal mucous membrane will also be a mode of entrance for infection. Menger demonstrated in cases of acute articular rheumatism following tonsillitis, bacteria under the epithelium of the tonsil mucous membrane; but he found them likewise in the mucous membrane of the neighboring tissues, and in these tissues they were deeper and much further advanced from the surface to the capillaries.

The tonsil is supposed by many to be quite frequently the mode of entrance for many diseases, notable among them tuberculosis. Dieulafoy took the tonsils and adenoids of ninety-six individuals, cut out the centers of these tonsils and adenoids, and put them under the skin of guinea pigs. He claims to have obtained as a result of the tonsils, tubercular infection in 12 per cent of the animals; and as a result of the adenoids, tubercular infection in 20 per cent of the animals. A number of men have repeated these experiments and have failed to obtain any striking results, except from a negative point of view. A year ago the writer repeated these experiments. Tonsils and adenoids were selected from fifty children —some of whom were tubercular and others of whom had a tubercular family history; these tonsils and adenoids were put under the skin of guinea pigs, as was done in Dieulafoy's cases. Of these, six died of streptococcus infection within a week, three died of pneumonia, only one showed any tubercular lesion, and the rest were prospering very notably when last seen, at the end of three months.

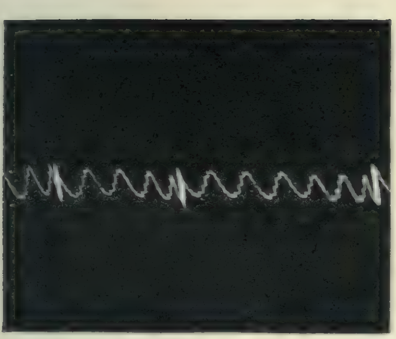
It would seem from the foregoing that the tonsil under ordinary conditions is not a portal of infection any more than any other tissue of the body, but rather a part of the protective mechanism of the system. It is also true, however, that when in a diseased condition, like any other diseased tissue of the body, it may become a mode of entrance for infection. This, however, is dependent upon other factors also. These factors are, first, the particular resistance of the individual; and second, upon the degree of virulence and pathogenicity of the bacteria concerned in each infection. Further, the tonsil, from its strategic position in the throat, is in the pathway of infection coming from many sources, and is perhaps in that way more open to infection than most of the body tissues.

In conclusion, it is fair to assume that the tonsil has a function, but probably only in the early years of life; it is also fair to assume that there is other tissue in the body, as for instance the lymph glands, which have the same function;

this function is mainly protective, however it may be explained. Removal of the tonsil, therefore, does not remove from the body any organ or tissue absolutely necessary to the system, but, if not diseased, its removal has left the individual with one defense less.

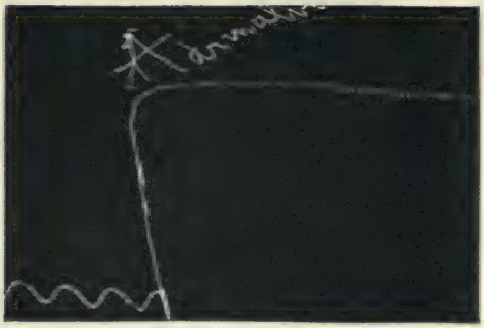
BIBLIOGRAPHY.

Gray's Anatomy.
Thomson: Diseases of the Nose and Throat.
Ballenger: Diseases of the Nose, Throat and Ear.
Moritz Schmidt and Edmund Meyer: Die Krankheiten der oberen Luftwege, 4 Auflage.
Dieulafoy: Textbook of Medicine, Vol. 1. Appleton, 1911.
Schoeneman: Fraenkel's Archives, Bd. XXIV, 1909.
Brieger and Goerke: Arch. für Laryngologie, Bd. XIX, No. 2.
Knight and Bryant: Diseases of the Nose, Throat and Ear, 2d Edition, Blakiston.
Wright, Jonathan: New York Medical Journal, August 8, 1909.
Wright, George: Boston Medical and Surgical Journal, May 20, 1909.
Ashhurst: Am. Jour. Med. Sciences, July, 1909.
Swain: Annals of Otology, Rhinology and Laryngology, September, 1911.
Masini: Clinica Medica Italiana, May, 1898.
Scheier: Berl. Laryng. Ges., 1903.
Pugnat: Belato-Laryngol. Gesell., Brussell, 1903.



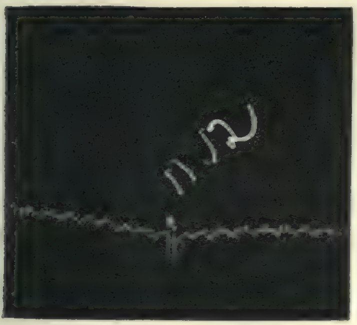
Tonsil Extracts.

Salt solution extract injected. Note the absence of change in curve.



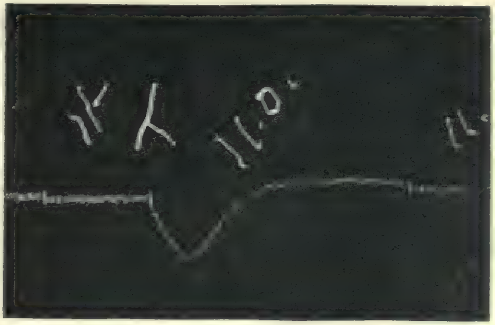
Adrenal Extract.

Note great rise in curve caused by injection of adrenalin solution 1/10,000 in very small amount.



Copper Chlorid Extract.

Note lack of change in curve from normal after injection of tonsillar extract made by treatment of tissue with copper chlorid, and then removing the copper.



Extract Not Copper-free.

Note the fall in blood pressure caused by the presence of copper in the extract. When same solution is made free of copper the curve remains normal.

X.

SUBMUCOUS RESECTION OF THE NASAL SEPTUM IN THE SEMIRECUMBENT POSITION.

By William R. Butt, M. D.,

Philadelphia.

To secure reasonably uniform success in all types of septal deflections by submucous resection, the following are essentials:

First—A knowledge of the anatomy of the septum—particularly that gained from studying it in relation to its embryologic development. (See a previous paper.)*

Second—An operating position and anesthesia which secure comfort to the patient and at the same time ease of manipulation to the operator; and in operating under cocain anesthesia, endeavoring to eliminate the personal equation of the patient as far as possible.

Third—Detailed methods of technic to overcome the mechanical difficulties.

The semirecumbent position, in my opinion, is ideal for nasal and oral operations done under local anesthetics. The position comprehends the patient lying on a table with the back raised at an angle of 45 degrees, and with the headpiece of the table made adjustable to a position comfortable alike to the patient and the operator. An electric bulb on an adjustable bracket or placed in a proper position on the wall, and an ordinary head mirror as a source of light, have proven most satisfactory, though a Freeman or Kirstein light have some advantages.

The toxic systemic effects of cocain and adrenalin with the patient in this position are incomparably less than those produced by these drugs on a patient in a sitting position. Since using this position, and the method of anesthesia to be described, during the year past, the operation has not been interrupted by the patient in any case and there has been practically no complaint of pain or discomfort. The patient's head rests in a steady position against the table; he is at rest and comfortable and there is no necessity for hurry. The patient does not complain of stiff neck or grow weary, as he does from the strained positions made necessary in doing the operation in the sitting position. The headpiece of the table is raised to a position in which the operator, while standing, can work comfortably without stooping. If an assistant is employed, he can work at the opposite side of the table without interfering with the operator. The patient's head is kept rather to the side of the table from which the operator works. I now have an Allison table which permits this position for use in the office, and with the arrangements shown in the illustration the operation can be done without assistance. Two days' stay in the hospital, however, is much preferable when feasible.

---

*"Some Considerations in Reference to the Nasal Septum." The Laryngoscope, December, 1912.

The following method of local anesthesia has been highly satisfactory: First, both entire nasal fossæ are wiped over roughly once or twice with cotton wound applicators dipped in ten per cent cocain to produce general superficial insensibility throughout to further manipulation. In a few minutes, cotton tipped applicators dipped in full strength adrenalin are dipped, after the excessive moisture has been removed, in powdered cocain. This socalled "cocain-adrenalin mud" of Freer is massaged thoroughly into the entire septum on both sides. Two or three applicatorsful suffice. Then one per cent cocain in 1-10,000 adrenalin (which is made up roughly as desired by adding three drops of adrenalin to thirty drops of one per cent cocain solution) is injected hypodermically along the line where the incision is to be made and along the nasal floor. This last hypodermic injection secures anesthesia along the base of the ridge, which is usually a painful spot, and also in the skin margins, which are not easily reached by cocain which is rubbed in.

These three procedures usually secure entire anesthesia and blanching of the entire septum, so that no further applications are needed. However, after the first incision is made, if there is bleeding, full strength adrenalin solution is rubbed in the cut; but I believe that the success of the operation depends considerably upon securing satisfactory anesthesia and ischemia at the outset before making the primary incision. This requires about fifteen minutes.

Since hearing of the death of a patient instantly following the hypodermic injection of ten minims of 1-1000 adrenalin into the nasal septum, reported by Dr. Freudenthal, I have never used adrenalin solution full strength hypodermically. I have myself observed severe symptoms of shock following the hypodermic administration of full strength adrenalin into the septum—despite the fact that this drug is recommended for combating shock.

The following details of technic are those which I have adopted from various sources: I make the incision described by Yankauer—usually on the side of the septum, which is convex anteriorly. An incision in the patient's left nostril has the advantage that the right hand is more easily used for the operating instrument, while a finger of the left hand can be inserted in the opposite nostril or used in holding the speculum, etc. If the deflection is very much to the right, however, the incision is made on that side, and in this case by bringing the left hand over the patient's face (while standing on the patient's right) the right hand is still free to do the actual dissecting, and a finger of the left hand may be inserted in the patient's left nostril.

The incision begins at the highest part of the septum in front of the deflection, and is carried down to the floor of the nose and partly across the nasal floor, practically into the skin of the vestibule. This incision also seems to me ideal. If each nasal fossa be represented as a right angled triangle, with the septum as the upright limb, then by this incision we lift the mucoperiosteal flap away from the vertical and horizontal limbs, allowing the loose flap to be pushed against the outer nasal wall (which represents the hypothenuse of the triangle). This gives a complete exposure of the septum and room for instruments without any stretching of the flap, as is necessary in a simple buttonhole incision. After the operation there is no retraction of this kind of a flap. The cut of the primary incision must be deep enough to go through the periosteum and perichondrium, but not necessarily at one stroke—the line of the incision may be gone over a second or third time, to make sure that it is down to the cartilage and bone. A very helpful procedure in ascertaining whether the perichondrium has been cut through is to take the side of the point of the knife and scrape it along the line of incision until the cut edge of the perichondrium is seen. Then the cartilage is separated from the mucoperichondrium as far down as the vomer ridge, beyond which dull dissection cannot be carried, because the periosteum of the vomer here crosses from one side of the septum to the other. Freer's round knife and a sharp pointed spoon curette are useful in carrying the primary incision completely down to the bone along the lower part of the septum and into the nasal vestibule. Beginning now again in the primary incision near the nasal floor beneath the ridge, taking advantage of the fact that the periosteum is easily separated here, the thin dull Freer dissector is inserted under the periosteum here at the floor and worked up towards the top of the vomer ridge.

Now we have the entire flap lifted up from the convex side of the septum except at the single line along the vomer ridge, where it can be seen as a longitudinal band passing over the crest of the ridge. This is easily cut by a sharp dissector from below, often also assisted by scraping with a sharp spoon curette from above, with the cutting edge turned in toward the bone.

In getting through to the other side of the septum a little furrow is first scraped with the curette or point of the knife used sideways, a little deeper in the nose than the incision through the mucous membrane. Into this furrow the semi-convex end of a small (Potts') dissector is inserted with the convex side turned toward the opposite nostril, using a finger in the opposite nostril as a guide. With this technic of incising the cartilage a perforation is seldom made at this point. The cartilage is freed on the opposite side down to the vomer ridge, and the necessary cartilage is removed by swivel knife and flat forceps. The adherent point on this side along the crest of the ridge is freed by cutting with the sharp dissector from above and scraping with a sharp spoon curette turned in toward the bone, as described for the other side, and the periosteum is separated down to the nasal floor. Both flaps are now entirely free, and the cartilaginous septum removed. The bony ridge is cut along the floor with the Hurd forceps (which avoids rough fracturing) and the bone bitten out with small forceps—the extreme anterior part being bitten out by holding the forceps perpendicular to the nasal floor; and sometimes by hooking the beaked end of the mastoid spoon curette around it and fracturing it forward.

The necessary portion of the perpendicular plate of the ethmoid is removed by fracturing it off with small strong double curette beaked forceps, an attempt being made to have the lines of fracture such that they will not be carried up into the cribriform plate. A bone cutting forceps is probably safer for removing the perpendicular plate, but those forceps on the market are too large and clumsy. That it is possible to open into the cribriform plate by the ordinary septum forceps used carelessly, I have demonstrated to myself on the cadaver. The suggestion has been made that opening the cribriform plate, though undesirable, is probably little more dangerous than exposing the dura in a mastoid operation, which is seldom followed by serious consequences.

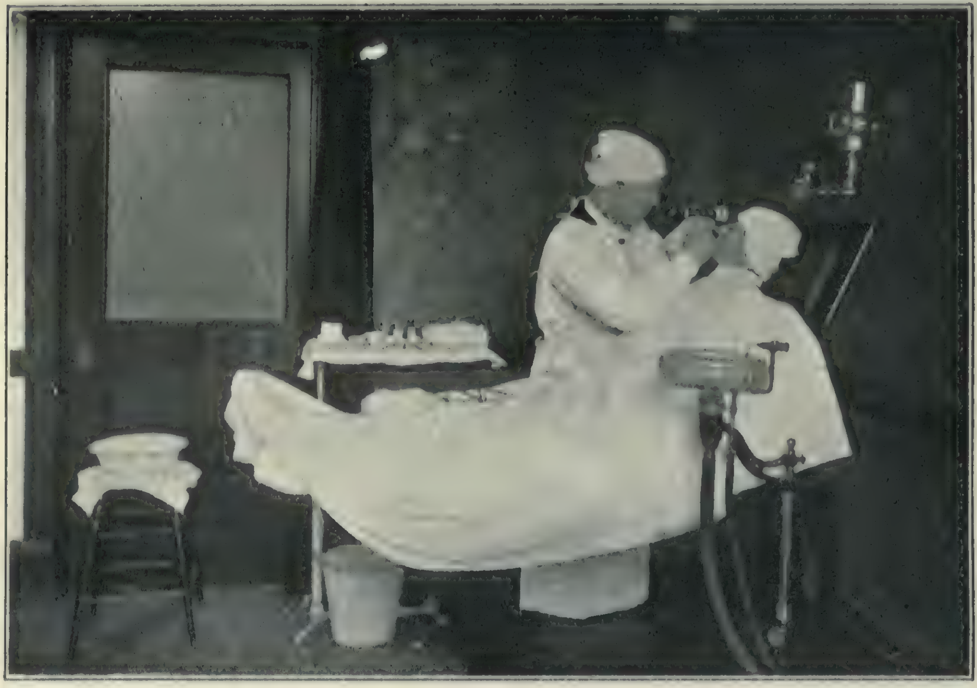
I have used both cotton splints and gauze packing, and have had hematomata of the septum following both. These I believe were due to the fact that the packing was too tightly placed over the lower and anterior parts of the septum and over the mucous membrane incision, preventing escape of what blood might exude between the flaps after the operation. The ultimate results of these hematomata when opened and drained were practically as good in a couple of weeks as though none had occurred. However, in the first two cases of this kind encountered, these blood clots were not cleaned out, and when the patients passed from observation the hematomata had apparently organized and the nasal fossæ were more obstructed than before operated on.

At present I use the cotton splints, packing both sides and placing them rather high, not bringing them farther anterior in the nose than the line of the primary incision in the mucous membrane. The packing is removed in from eighteen to twenty-four hours.

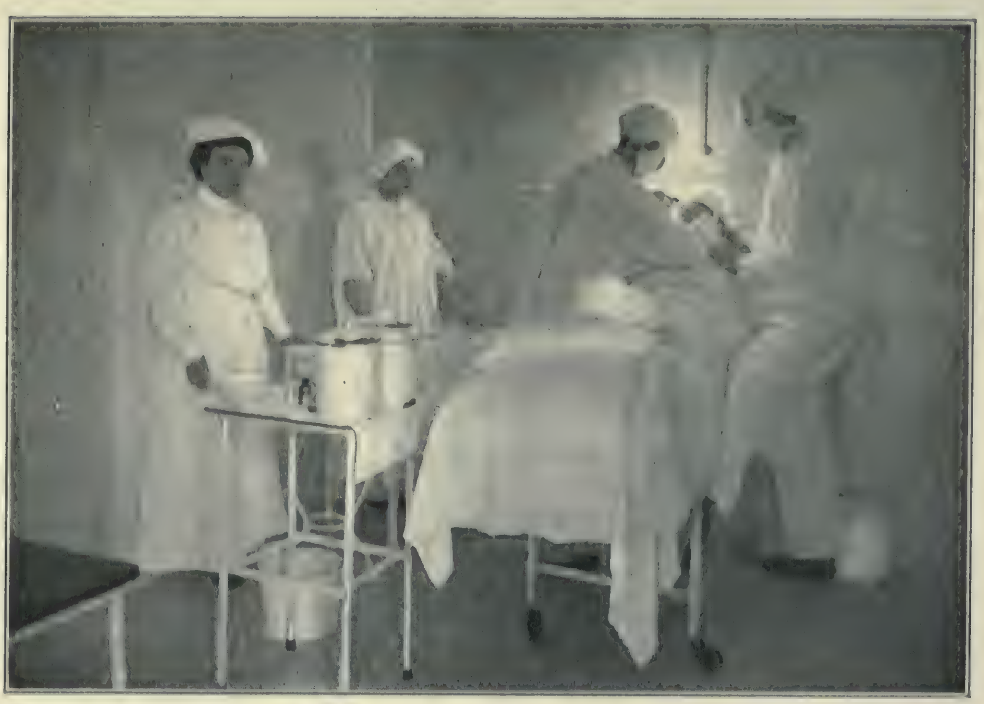
In two cases in young persons, where a general anesthetic was thought necessary, they were etherized lightly, then the septum was prepared as for local anesthesia, only sufficient ether being given to keep the patient asleep, depending upon the local anesthesia to abolish sensation. If it were safe to use adrenalin hypodermically in unlimited quantity, the operation would be much easier under general anesthetics. In one of these two cases under ether the bleeding was well controlled and the operation easily done—in the other there was considerable bleeding, and the work had to be done largely by the sense of touch, which resulted in the loss of considerable mucous membrane on one side, but the septal deformity was satisfactorily corrected, and there was no perforation. In this case the pharyngeal tonsil was also removed before putting in the nasal packing. In another case, after the submucous resection was done under cocain anesthesia, ether anesthesia was used to remove the adenoid mass.

In operating under general anesthesia, the semirecumbent position has the disadvantage that mucus and blood, and also the tongue, tend to fall backward and obstruct the breathing. These tendencies can be overcome by inserting a postnasal tampon and keeping the tongue held forward with tongue forceps.

In conclusion, I express indebtedness to Dr. Wood, Dr. Babbitt and Dr. Butler for hospital opportunities for doing part of this work.



Office Arrangement for Septum Operation.



Hospital Arrangement for Septum Operation.

XI.

TREATMENT OF DEAFNESS BY REEDUCATING THE HEARING.

By Dr. Maurice,

Paris.

Three elements come into play in the mechanism of audition —the muscles, the nerves, the organs of transmission. If one of these elements is damaged, the hearing is affected.

The treatment is determined by these conditions. Until recently we have been very much occupied with the tympanic organs, the organs of transmission, the tympanum and ossicles, but too little with the labyrinth and the organ of Corti:

Let it be understood that I am not discussing absolute deafness, but the generality of cases, which includes all those persons attacked with hardness of hearing, more or lass accentuated, even quite advanced, but not complete deafness. So, we do not treat deafness, but impaired hearing. To reawaken a sense something must remain; we do not create, we invigorate.

I will not speak of the well known methods of retarding or lessening deafness, such as general treatment of the causal diathesis, nasal treatment, catheterization of the tube, bougieing, mobilization of the tympanum and ossicles, etc. I have been looking for a method of perfecting the results thus attained.

I have been preceded in this line by Urbantschitsch, Rousselot, Natier, Marage, Surguet, Villot and others, and a study of their methods has helped in establishing my conclusions.

The sound wave is the normal functional excitant of the ear. Its vibrations make the muscles of the middle ear tense, the hammer and stirrup to listen or to protect themselves. It keeps up the free articulatory play of the ossicles. It strikes the organ of Corti and excites its functioning. The essence of the problem consists, then, in regulating, dosing, and exactly measuring the quantity and quality of sound wave necessary for this physiologic stimulus.

The method of Dr. Villot, which consists in exercises with an ear trumpet, is worthy of interest. It would be the method for a patient who has time to wait for improvement; it would not be a rapid, intensive reeducation.

To obtain the maximum of intensive reeducation, which I am discussing, we must have an apparatus for producing sound waves with certain desired characters. The tonality should vary at will and comprise the entire zone of the human voice.

The intensity of the sound should be modified according to the sensitiveness of the patient. One with dysacusia requires less intense sounds; another will stand the maximum.

Regularity of emission is quite important, since the intensity having been determined, we then have only to vary the tone.

Facility must be had for regulating the sounds reaching the ears, each independent of the other.

It is necessary that the vibrations produce a genuine sound massage of the organ, such as gives the ear a sense of titillation.

The principal note supercharged with numerous harmonics should give much resemblance to the human voice.

We could hardly ask more from an apparatus for reeducating the hearing, unless it is the telephonic transmission of the sounds obtained. In fact, the vibrating plate, placed close to the ear, imprisons between itself and the drum a cushion of air which transmits the entire vibration to that organ.

I am particularly pleased to state that I have succeeded in constructing an apparatus which fulfills the above conditions. I have christened it the "Kinesiphone," because of the vibratory massage effect combined with the emission of sounds.

In my apparatus the sounds are due to interruptions of the current in an electric resistance circuit on the phone wire, permitting regulation of the intensity. Tonality is obtained by an elastic vibrating plate, which augments the number of vibrations. To get a greater variety of sounds I have three plates, corresponding to low, medium, and high. The chords are due to the phenomena of self-induction which take place in the circuit.

While we are on the subject of sense reeducation I want to say that I highly esteem Dr. Gernet's method, which consists in giving the muscles of the middle ear a progressive stimulation, a functional reeducation which is of the greatest utility for the end which we seek. I advise it systematically for all the patients that I treat, and especially for those who have retardation of auditory accommodation.

The best way of demonstrating the worth of a method is to show cases. From the large number which I have I will cite the principal ones. I will select them from all classes of deafness, for I affirm and will prove that the reeducation treatment applies to all forms; sclerosis of the tympanum (cases II and III), of the labyrinth (cases IV, V and VI), primary or rhinogenic sclerosis (case III), dry cicatricial otitis with atrophy of drum and ossicles (case I), etc.

The improvement obtained has been determined by measuring for the watch, whispered voice and spoken voice. The multitude of measurements eliminates error.

For the voice test I pronounce at the end of expiration words so chosen that their enunciation will not be forced or involuntarily amplified. The words which I use fall into two classes: low, isosonorous words and high isosonorous words. This classification comes from experienced phoneticians, such as Rousselot, Hermann, Zwaardemacker, Marage, and others.

The low note is generally less well heard by sclerosed thickened tympani, while the high note is less easily heard in labyrinth trouble with normal tympanum.

In the following reports I have taken the mean between the high and low, unless otherwise indicated.

Spoken voice is indicated by a straight line (————), whispered voice by a series of dashes (— — — — —), and the watch by dots (. . . . . .).

CASE I.

Miss G., 18 years, neglected otorrhea had caused loss ot tympanum and ossicles. I was able to dry the ears, but not to affect the hearing. Reeducative treatment alone has given a most interesting result.

WATCH.

	Right Ear.	Left Ear.
Before December 7, 1911	7 c. m.	3 c. m.
After six sittings, February 21, 1912	42 c. m.	8 c. m.

WHISPERED VOICE.

	Right Ear. Isosonal Words.		Left Ear. Isosonal Words.	
	Low.	High.	Low.	High.
Before	0.4 m.	0.8	0.12	0.35
After	7. m.	12.	5.	6.5

VOICE.

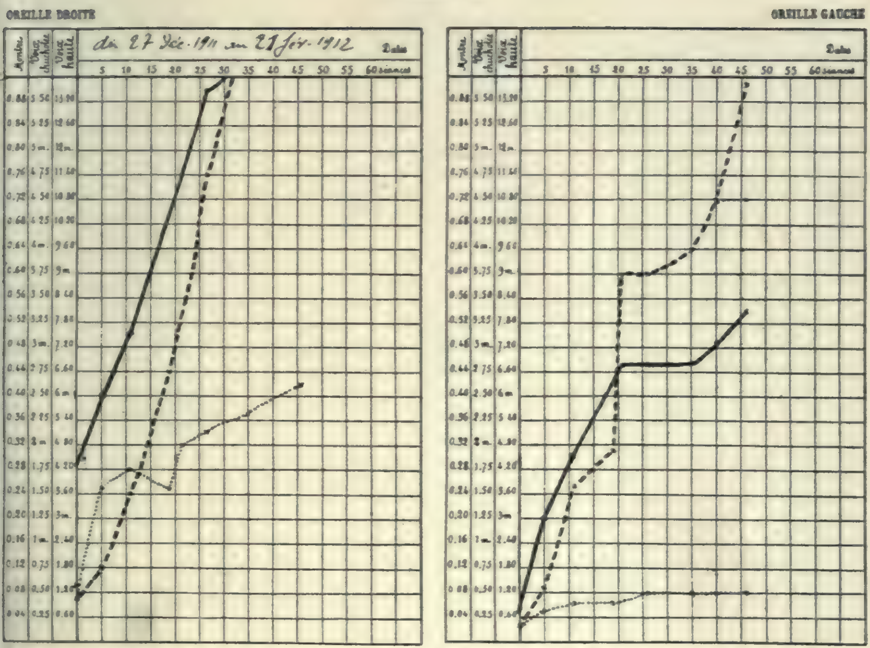
	Right Ear. Isosonal Words.		Left Ear. Isosonal Words.	
	Low.	High.	Low.	High.
Before	3.5 m.	5.	0.4	1.2
After	15. m.	15.+	6.5	9.

I defy any other method to produce such a result. The success can only be explained by mobilization of the cicatrices obstructing the labyrinthine windows, and by excitation of the organ of Corti which, however, was in good condition prior to treatment.

This young lady experienced transitory relief, before reeducation, from destroying the cicatricial membrane obstructing the round window. This slight improvement disappeared in two or three days, when the cicatrix reformed.

When seen April 19th she heard very well, without any regression.

CASE I.


OREILLE DROITE
OREILLE GAUCHE
Montre
Voix chuchotée
Voix haute
du 27 Déc. 1911 au 27 Fév. 1912
Date
5
10
15
20
25
30
35
40
45
50
55
60 séances
0.88 5.50 13.20
0.84 5.25 12.60
0.80 5m. 12m.
0.76 4.75 11.40
0.72 4.50 10.80
0.68 4.25 10.20
0.64 4m. 9.60
0.60 3.75 9m.
0.56 3.50 8.40
0.52 3.25 7.80
0.48 3m. 7.20
0.44 2.75 6.60
0.40 2.50 6m.
0.36 2.25 5.40
0.32 2m. 4.80
0.28 1.75 4.20
0.24 1.50 3.60
0.20 1.25 3m.
0.16 1m. 2.40
0.12 0.75 1.80
0.08 0.50 1.20
0.04 0.25 0.60

Case II.

Mr. D., aged 33, deaf since the age of 15, so that study was prevented. Diagnosis: Tympanic sclerosis, labyrinth unaffected. Previously treated by others and myself in the usual ways, especially catheterization, without result.

WATCH.

	Right Ear.	Left Ear.
December 21, 1911, before reeducation..........	1. c.	1. c.
January 30, 1912, after 69 treatments............	19. c.	14. c.

WHISPERED VOICE.

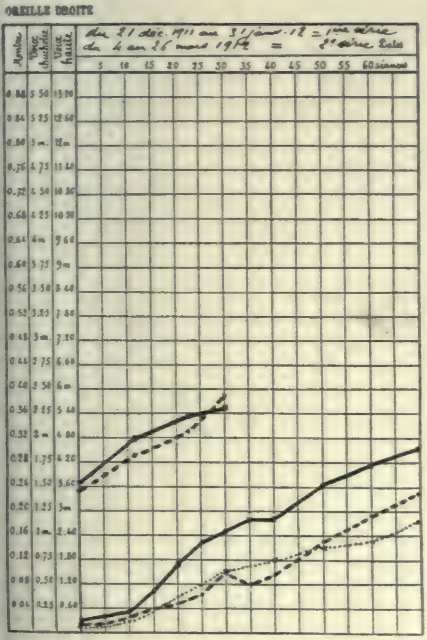
	Right Ear. Isosonal Words.		Left Ear. Isosonal Words.	
	Low.	High.	Low.	High.
Before	Ad aud.	0.18 m.	Ad aud.	0.18 m.
After	0.40 m.	2.50 m.	0.35 m.	3.20 m.

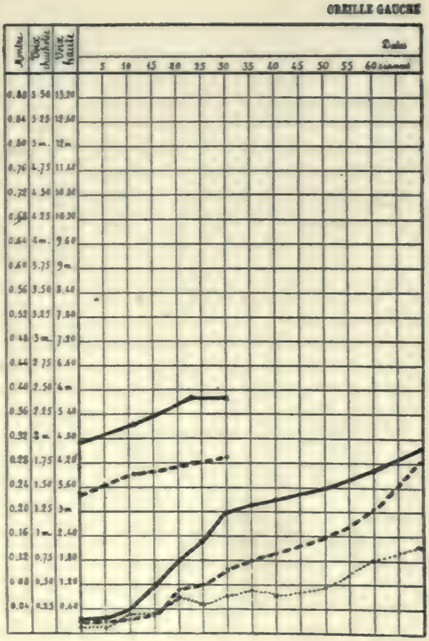
VOICE.

	Right Ear. Isosonal Words.		Left Ear. Isosonal Words.	
	Low.	High.	Low.	High.
Before	0.10 m.	0.60 m.	0.10 m.	0.60 m.
After	3. m.	6. m.	3. m.	6. m.

The result is particularly gratifying because of the duration of the trouble. The patient having demanded a quick cure, I give him thirty treatments from the 4th to the 26th of the month, which brought a little improvement, as may be seen on the chart (upper curve). As the patient lived in the province, I gave two treatments daily to gain time. The return of his hearing has enabled Mr. D. to change his business.

CASE II.


OREILLE DROITE
Montre
Voix chuchotée
Voix haute
du 21 déc. 1911 au 31 janv. 12 = 1re série
du 4 au 26 mars 1912 = 2e série Dates
5 10 15 20 25 30 35 40 45 50 55 60 séances
0.88 5.50 13.20
0.84 5.25 12.60
0.80 5m. 12m
0.76 4.75 11.40
0.72 4.50 10.80
0.68 4.25 10.20
0.64 4m 9.60
0.60 3.75 9m
0.56 3.50 8.40
0.52 3.25 7.80
0.48 3m 7.20
0.44 2.75 6.60
0.40 2.50 6m
0.36 2.25 5.40
0.32 2m 4.80
0.28 1.75 4.20
0.24 1.50 3.60
0.20 1.25 3m
0.16 1m 2.40
0.12 0.75 1.80
0.08 0.50 1.20
0.04 0.25 0.60


OREILLE GAUCHE
Montre
Voix chuchotée
Voix haute
Dates
5 10 15 20 25 30 35 40 45 50 55 60 séances
0.88 5.50 13.20
0.84 5.25 12.60
0.80 5m. 12m
0.76 4.75 11.40
0.72 4.50 10.80
0.68 4.25 10.20
0.64 4m. 9.60
0.60 3.75 9m
0.56 3.50 8.40
0.52 3.25 7.80
0.48 3m. 7.20
0.44 2.75 6.60
0.40 2.50 6m
0.36 2.25 5.40
0.32 2m 4.80
0.28 1.75 4.20
0.24 1.50 3.60
0.20 1.25 3m
0.16 1m 2.40
0.12 0.75 1.80
0.08 0.50 1.20
0.04 0.25 0.60

Case III.

My colleague, Dr. L., of Paris, aged 30, has tympanic sclerosis, probably rhinogenic, for the tube is atresic and he is subject to coryzas. Treated by me for six months he got no results, in spite of a thorough nasal treatment, insufflation of hot air, and vibratory massage. His condition grew worse from day to day until he could hear only with difficulty. I then tried reeducation.

WHISPERED VOICE.

	Right Ear. Isosonal Words.		Left Ear. Isosonal Words.	
Dec. 22, 1911—	Low.	High.	Low.	High.
Before reeducation	0.30 m.	0.80 m.	1. m.	7. m.
Feb. 7, 1912—				
After treatment	6. m.	7.50 m.	12. m.	15. +m.

VOICE.

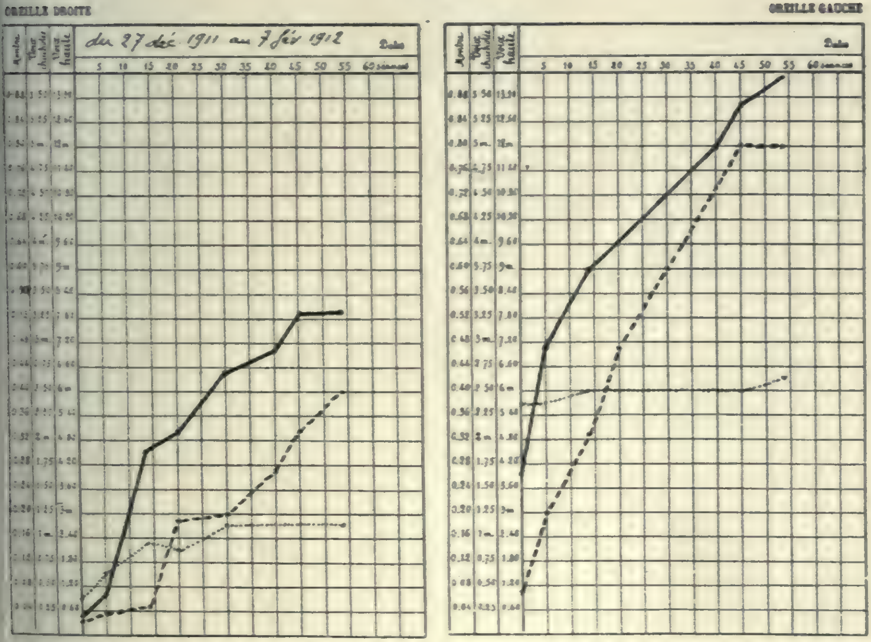
	Right Ear. Isosonal Words.		Left Ear. Isosonal Words.	
	Low.	High.	Low.	High.
Before	0.40 m.	1. m.	4. m.	8. m.
After	8. m.	13. m.	14. m.+	15. m.

Right hearing for the watch has increased in the same time from 6 cent. to 18, and the left from 28 to 42 only. Words "ou" and "on," not perceived by the right ear, were heard at the end of treatment from 1.75 to 2. m. My colleague can really hear very well. I was particularly gratified with this result in a physician who could himself estimate the benefits of the method.

Seen March 22, 1912, the amelioration was maintained in spite of a cold which the patient then had. Audition for the watch had even increased on the left from 42 to 48 cm., although it had remained almost stationary during treatment.

The chart is traced for isosonal low words. Audition for whispered words is shown in the third column (that for voice), for whisper being heard almost as well as voice, the left tracing would have gone out after the eighteenth sitting if it had been inserted in its regular place (second column).

CASE III.


OREILLE DROITE
OREILLE GAUCHE
du 27 déc 1911 au 7 fév 1912
Date
Montre
Voix chuchotée
Voix haute
5
10
15
20
25
30
35
40
45
50
55
60 séances
0.88 5.50 13.20
0.84 5.25 12.60
0.80 5m 12m
0.76 4.75 11.40
0.72 4.50 10.80
0.68 4.25 10.20
0.64 4m 9.60
0.60 3.75 9m
0.56 3.50 8.40
0.52 3.25 7.80
0.48 3m 7.20
0.44 2.75 6.60
0.40 2.50 6m
0.36 2.25 5.40
0.32 2m 4.80
0.28 1.75 4.20
0.24 1.50 3.60
0.20 1.25 3m
0.16 1m 2.40
0.12 0.75 1.80
0.08 0.50 1.20
0.04 0.25 0.60

Case IV.

Mme. G., aged 44, sent to me by Dr. Hansen, of Paris. This lady could not hold conversation at a distance of 1.5 m. at the beginning of treatment, as Dr. Hansen and I confirmed. She had otitic adhesions with discharges from the labyrinth, and this trouble antedated three years. The drum was retracted, so I commenced by catheterizations, which were continued for a month or so. Her condition remaining the same, I attempted reeducation, after her hearing had been exactly measured.

WATCH.

	Right Ear.	Left Ear.
Dec. 22, 1911, before reeducation	2. c.	.35 c.
Jan. 18, 1912, after 24 treatments	22. c.	.40 c.

WHISPERED VOICE.

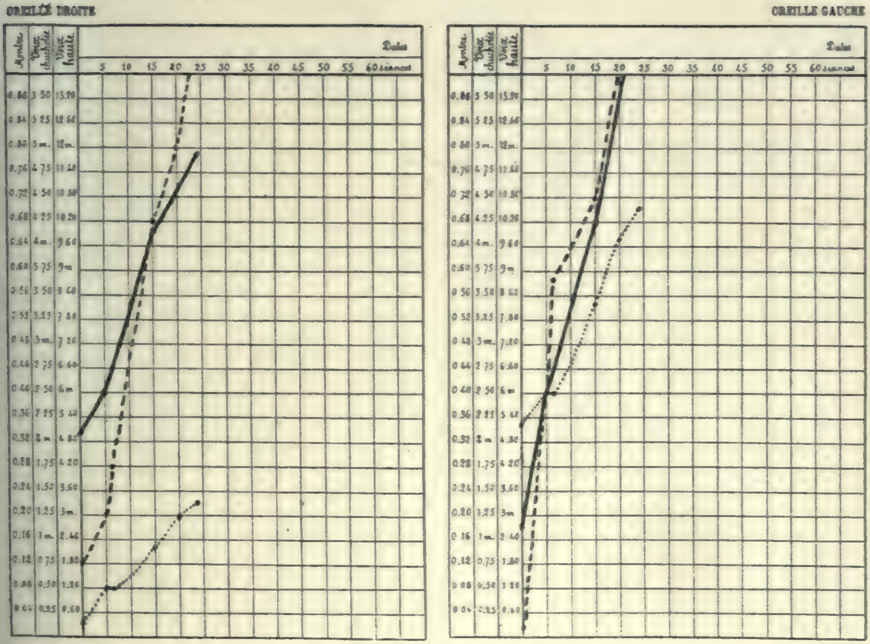
	Right Ear.		Left Ear.	
	Low Tones.	High Tones.	Low Tones.	High Tones.
Before	0. m.	35. m.	0.15 c.	0.01 c.
After	4. m.	50. m.	5. m.	12. m.

VOICE.

	Right Ear.		Left Ear.	
	Low Tones.	High Tones.	Low Tones.	High Tones.
Before	4. m.	6. m.	1.50 m.	4. m.
After	12. m.	12. m.	15. m.	15. m.

A rather stubborn eczema of the canal was very much improved by the kinesiphonic treatment, and that I attribute to the effect of the vibration. The eczema of the canal produced, by irritation from a branch of the pneumogastric, a morning cough which disappeared entirely after a few treatments. I desire to call attention to the hearing in the left ear of the low tones of the whispered voice, which has increased from 1 c. to 12 m. The patient when last seen had continued improving.

CASE IV.


OREILLE DROITE
OREILLE GAUCHE
Montre
Voix chuchotée
Voix haute
Dates
5 10 15 20 25 30 35 40 45 50 55 60 séances
0.88 5 50 13.20
0.84 5 25 12.60
0.80 5 m. 12 m.
0.76 4 75 11.40
0.72 4 50 10.80
0.68 4 25 10.20
0.64 4 m. 9.60
0.60 3 75 9 m.
0.56 3 50 8.40
0.52 3 25 7.80
0.48 3 m. 7.20
0.44 2 75 6.60
0.40 2 50 6 m
0.36 2 25 5.40
0.32 2 m 4.80
0.28 1.75 4.20
0.24 1.50 3.60
0.20 1.25 3 m.
0.16 1 m. 2.40
0.12 0.75 1.80
0.08 0.50 1.20
0.04 0.25 0.60

CASE V.

Mr. Ar., 52 years, found that his hearing, especially in the left ear, was gradually growing worse. I found, after examination, adhesive otitis, with involvement of the labyrinth; the drum was relatively large, and catheterizations had had no good effect. The involvement from the labyrinth discouraged me, but nevertheless I submit the results.

WATCH.

	Right Ear.	Left Ear.
December 22, 1911	10. c.	2. c.
January 10, 1912 (26 treatments)	35. c.	11. c.

WHISPERED VOICE.

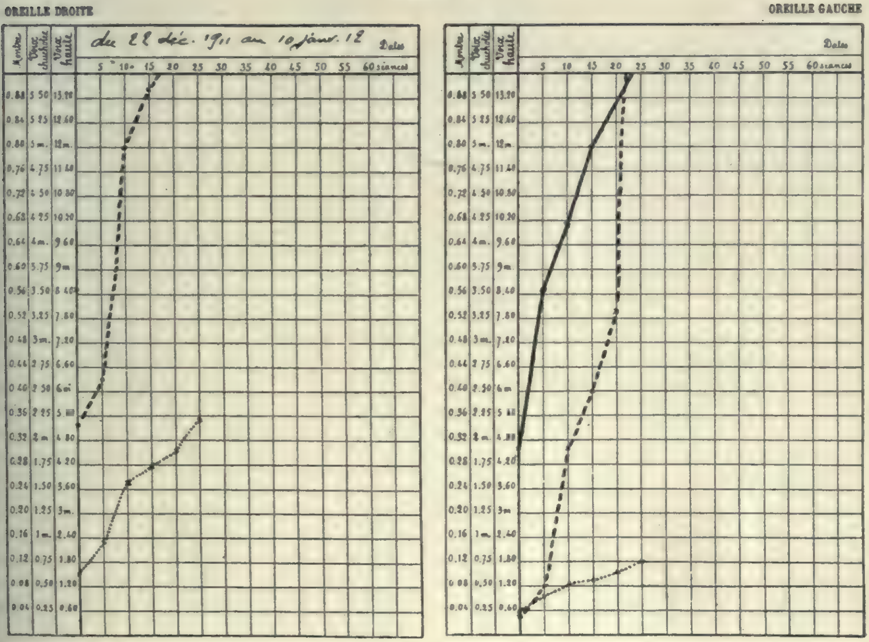
	Right Ear.		Left Ear.	
	Low Tones.	High Tones.	Low Tones.	High Tones.
Before	1. m. 80.	2. m. 50.	0. m. 30.	0. m. 10.
After	8. m.	13. m.	10. m.	8. m. 50.

VOICE.

Right Ear.	Left Ear.	
	Low Tones.	High Tones.
Impossible to measure because of its relatively good condition.	5 m.	2 m.
	Room too small to measure improvement. Call it 15 m.+	

It is the improvement in the whispered voice of which I shall make special mention. The patient's hearing was measured after the twenty-sixth treatment, as I stopped there, considering the result sufficient. When spoken to, Mr. A. made no distinction between the hearing of either ear, while before treatment he was obliged to place the speaker to his right ear.

CASE V.


OREILLE DROITE
OREILLE GAUCHE
Montre
Voix chuchotée
Voix haute
du 22 déc. 1911 au 10 janv. 12
Dates
5 10 15 20 25 30 35 40 45 50 55 60 séances
0.88 5 50 13.20
0.84 5 25 12.60
0.80 5m. 12m.
0.76 4.75 11.40
0.72 4.50 10.80
0.68 4.25 10.20
0.64 4m. 9.60
0.60 3.75 9m.
0.56 3.50 8.40
0.52 3.25 7.80
0.48 3m. 7.20
0.44 2.75 6.60
0.40 2.50 6m
0.36 2.25 5.40
0.32 2m. 4.80
0.28 1.75 4.20
0.24 1.50 3.60
0.20 1.25 3m.
0.16 1m. 2.40
0.12 0.75 1.80
0.08 0.50 1.20
0.04 0.25 0.60

Case VI.

Mr. U., fifty-five years old, was treated in 1908 by Dr. Archambaut for labyrinthic deafness which seemed to increase. He was hard of hearing from childhood; his father and two uncles were deaf. His deafness had increased very suddenly, as the result, he said, of a kiss on the ear. I thought this a special case, although the mercurial treatment had been of no avail. The catheterizations Dr. G— and I attempted did not help his hearing.

WHISPERED VOICE.

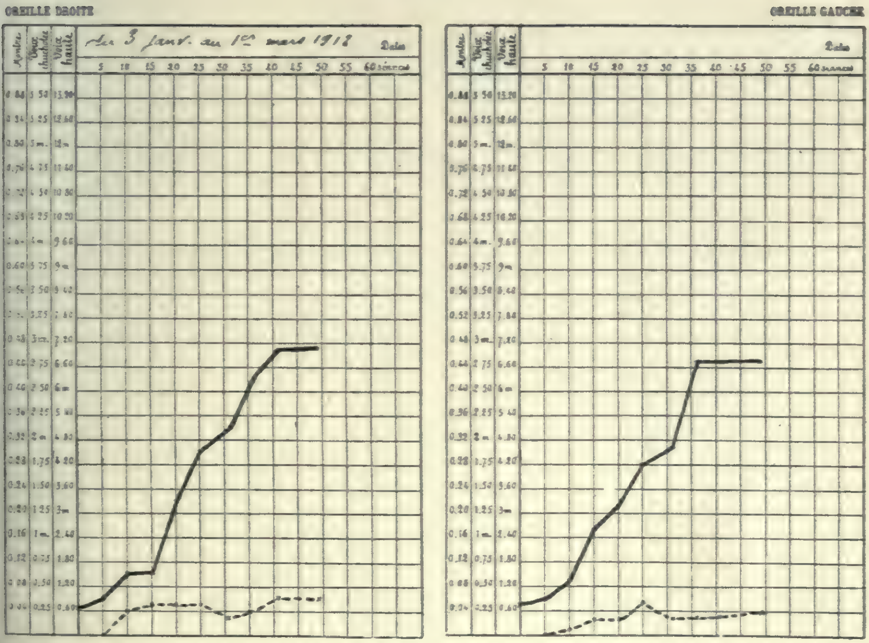
	Right Ear.		Left Ear.	
	Isosonal Words.		Isosonal Words.	
Jan. 3, 1912—	Low.	High.	Low.	High.
Before reeducation	Not heard	Not heard	Not heard	Not heard
March 1, 1912—				
After 50 treatments	0 m. 30	0 m. 25	0 m. 25	0 m. 25

VOICE.

	Right Ear.		Left Ear.	
	Isosonal Words.		Isosonal Words.	
	Low.	High.	Low.	High.
Before	0 m. 60	0 m. 80	0 m. 80	0 m. 80
After	7 m.	7 m.	6 m. 80	6 m.

The watch was not heard. The patient at first stood the acoustic exercises with difficulty; soon he very rapidly accustomed himself to the maximum.

CASE VI.


OREILLE DROITE
Montre
Voix chuchotée
Voix haute
du 3 janv. au 1er mars 1918
Date
5 10 15 20 25 30 35 40 45 50 55 60 séances
0.88 5.50 13.20
0.84 5.25 12.60
0.80 5m. 12m.
0.76 4.75 11.40
0.72 4.50 10.80
0.68 4.25 10.20
0.64 4m. 9.60
0.60 3.75 9m.
0.56 3.50 8.40
0.52 3.25 7.80
0.48 3m. 7.20
0.44 2.75 6.60
0.40 2.50 6m.
0.36 2.25 5.40
0.32 2m. 4.80
0.28 1.75 4.20
0.24 1.50 3.60
0.20 1.25 3m.
0.16 1m. 2.40
0.12 0.75 1.80
0.08 0.50 1.20
0.04 0.25 0.60
OREILLE GAUCHE
Montre
Voix chuchotée
Voix haute
Date
5 10 15 20 25 30 35 40 45 50 55 60 séances

The conclusion to be drawn from the observations above is that no other method is capable of producing these results, since all the patients noted had tried the standard treatments in use.

I am able to state that all kinds of deafness can be ameliorated, as well as one can claim that tuberculosis in the first stages is curable. There are no rules without exceptions, and in kinesiphonic reeducation, the exceptions are rare—one in thirty, from my actual observations.

In conclusion, I wish to attempt to explain the process of improvement and then of the cure.

Light massage produces, by mobilization of a physiologic sort, thickened tympanum and ankylosis of the ossicles. Pneumatic massage accomplishes very little, because these organs are intended to vibrate at a high degree of speed and not at the reduced speed of the masseur of Delstanche or the electromotor of Breitung.

Gentle vibration produces on the membranes of the drums of Narey molecular modifications which help them and which permit them by usage to make better inscriptions. These modifications are found in the case of a violin in which the sound is most harmonious after much training.

The tympanum should act in the same manner, after several treatments by reeducation.

The treatment succeeds in producing a vasodilatation visible to the otoscope, a vasodilatation whose fine result must be most useful to an organ almost destroyed. The return of the ceruminous secretion, the amelioration of certain dry eczemas of the canal, amply prove it and allow us to extend to the middle and inner ears what is ascertained in the external ear.

Powerful sounds given in the right way stimulate the indifferent hearing of a deaf person. Most deaf people are careless of hearing because they do not use their ears. Ultrarapid recoveries can hardly be explained except in this way. As soon as one does not hear the human voice well, he does not hear any longer because it tires him to listen; he isolates himself, as far as his hearing is concerned, until he relapses into a deafness more and more complete.

The stimulation of many fibers and cells of the organ of Corti by sounds befitting their number and their function can only reawaken an organ on the road to atrophy, to sclerosis, to functional paralysis.

Moreover, we can imagine that violent vibration favors the contact of cylindrical terminations and cells of the organ of Corti, as it produces between the carbon granules in a microphonic apparatus.

All these reasons can explain my results.

The improvement has been maintained and has the tendency to continue, for the patient, by himself, pursued his physiologic training. In the meanwhile he listens to noises and continues the reeducation.

If the cause of deafness persists in making the affection evolve, a loss of hearing is not manifested when the cause is not reached by reeducation; in that case it is good to attempt at the end of a while, a new one. In advising the patient to perform the exercises which Dr. Villot values, by means of an acoustic horn which is not measured, in most cases a non-receding amelioration is obtained.

The cases which I cite most strongly and which are quite controllable show unquestionably the foundation of the method and the hopes there are to expect of it. I am entirely of the opinion of my confreres who wish to judge de visu the truth of what I have put forth.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

Meeting of November 8, 1912.

Case Showing Graphite Stains in the Membrana Tympani.

Dr. A. P. Voislawsky: In February, 1911, J. C., four years of age, was brought to my office suffering from an acute discharge of the left ear, due to a traumatic injury of the membrana tympani, self-inflicted with a toy gun having a lead pencil as its projectile. The little patient placed the gun in the left auditory canal and pulled the trigger. The projectile entered the membrana tympani in the anterior inferior angle, and for six days there was an acute discharge which promptly yielded to treatment. One year and a half later, on examination, the hearing was normal, but at the point of entrance was a well marked graphite stain, showing that at the time of entrance some particles of the lead imbedded themselves in the membrana tympani.

A Case of Tumor in the Cerebellar Pontine Angle, Presenting Early Symptoms of Deafness and Vertigo. Preliminary Report of Autopsy Findings.

By John Alexander Robinson, M. D.

DISCUSSION.

Dr. Dixon said that it was difficult to state positively whether the case was an echinococcus cyst or not. Examination of the brain perhaps would have enabled one to be more positive. It certainly was a cyst, but what form it was not possible to state exactly.

Dr. Dench had seen the case with Dr. Robinson, and wished to emphasize the importance of a thorough otologic examination in all cases in which some lesion below the tentorium is suspected. He continually sees a number of cases in his service in the Neurological Institute, and finds the work of the otologist in making a careful examination to be of distinct value in localizing the exact position of the lesion. He would impress on all otologists the importance of a careful examination when tumors are suspected.

Dr. McKernon had been interested in observing how long these patients would live after simple decompression operations. He had seen two cases with Dr. Peterson which differed from Dr. Robinson's cases in having less hearing on affected sides, and tinnitus was more pronounced. One was operated upon by Dr. Cushing, a decompression operation being performed. The patient lived for eight months and four days after operation, suffering with a large brain hernia. The second died four and a half months after Dr. Cushing's operation, with a tremendous brain hernia present. These cases simply show how long on an everage such patients live after decompression operation.

Dr. Wilson asked for how long after the decompression operation did the optic neuritis remain cleared up.

Dr. McKernon replied that the neuritis improved for about six weeks, then increased with greater severity.

Some Systemic Factors in the Etiology of Progressive Deafness.

Dr. J. A. Stucky, in introducing this subject, deplored the fact that these conditions still stand impregnable to the attacks of the otologist, in that so little can be done for their relief and that so little is known of their inherent etiology, but extended the hope that in working in close relation with the internist and the neurologist, more can be known of the causation and more done to inhibit their progress, even though no cure is found.

Two classes of cases were cited, those of a nonexudative otitis media and those of otosclerosis, both resulting in progressive deafness. A definite pathology exists from each condition.

In the nonexudative otitis media there is little change in the membrana tympani, slight retraction, atrophy, opacity, or small calcareous deposits, the eustachian tube showing no change to account for the condition, and because of the progress of the deafness, it must be concluded that ankylosis is present in the ossicular joints and fenestral structures to a greater or less degree.

There may or may not be an associated nasopharyngeal condition. The etiology of these conditions still remains the shuttlecock of otology, but all agree that the cause is nonspecific, but due to some cause foreign to the ear itself. The drum membrane shows no deviation from normal, unless it be complicated by a nonexudative otitis.

In otosclerosis the disease begins in the bony capsule of the labyrinth, especially near the oval window, rarely at the fenestra rotunda. In the regions so affected the osseous tissues become vascular, giant cells and osteoblasts are present, forming overgrowth of spongy tissue, whereby the foot of the stapes and annular ligament are involved, with a resulting ankylosis. The function of the stapes is thereby interfered with or abolished.

Some victims of tuberculosis, syphilis, nephritis, rheumatism, organic heart and other circulatory conditions are affected secondarily with one or the other types of progressive deafness, due to circulatory changes in the ear, or to a hyperemia because of high arterial tension.

Most especially does the author emphasize in all cases treated by him, the close relation of disturbed metabolism and toxin absorption produced by improper food and drink, these toxins entering the blood and so causing disturbance in the ear, the point of least resistance in those so affected.

As food, drugs, toxin, and alcohol changed by the same elements that each victim so afflicted asserts his or her idiosyncrasy to the individual poison to which this susceptibility exists, so in brief the assumption that the changes are due, to a large extent, to autointoxication and other toxic agents, predicts that future findings will support this contention. Heredity, only in that like conditions, like food, apply to the child as they do to the parent.

Nonexudative otitis media and otosclerosis are related only symptomatically, those of the former often merging into those of the latter.

In the presence of slight tinnitus and vertigo, with a history of varying degrees of deafness aggravated by any systemic or psychic disturbance which unbalances in any way the circulatory apparatus of the labyrinth, accompanied by the slight pathologic changes in the membrana tympani referred to above, one must consider the probability of the nonexudative otitis media, provided they do not respond to inflation and local treatment and are not caused by some nasopharyngeal or eustachian tube derangement. By Weber's test the bone conduction is increased, except in old persons, when it will be found decreased in intensity. Most especially should one look for some systemic disturbance.

Subjectively the symptoms of otosclerosis are those above, but intense vertigo occurs less frequently and seldom in aggravated form. Bezold's symptom complex is found, increased bone conduction and negative Rinné with elevation of tone limit. The membrana tympani is practically unchanged, unless complicated by nonexudative otitis media.

The following case reports are incorporated:

Nonexudative otitis media, eleven cases, nine males, two females, varying from 23 to 68 years of age, with hearing 25 to 50 per cent below normal, following a gradual diminution for one to ten years.

These cases were improved 10 to 30 per cent by strict regime of diet and no alcohol, together with alterative remedies. Occasional exacerbation occurs when overindulgence in eating or drinking occurs.

In seven cases of otosclerosis, three males and four females, five of which cases were seen by several prominent otologists and a very unfavorable prognosis given, especially those in whom an hereditary element existed, the ages ranged from 22 to 47 years, with 10 to 35 per cent loss of hearing power. By the same treatment three regained 15 to 20 per cent, and the other four, who were worse and at more advanced age, regained 2 to 5 per cent. The diagnosis in both of these series of cases was made after careful and frequent observations.

Just what systemic derangements cause progressive deafness, the author cannot say, but places rheumatism and arteriosclerosis, from whatever cause, as most important.

All cases were much aggravated by any disturbance of metabolism, no matter how brought about.

The treatment of progressive deafness advocated by the author is, briefly: assist elimination, get rid of any toxic substance by stopping intestinal absorption of toxin, as nearly as possible preserve a normal tissue metabolism.

To be most emphasized is the fact that the two types of progressive deafness can be differentiated, though closely related. Whichever are intensely aggravated by any disturbance of metabolism and which can be improved, stayed in their progress and often in a marked degree relieved by careful and persistent treatment by use of a careful dietary and alterative treatment.

DISCUSSION.

Dr. McKernon said that he did not feel qualified to discuss Dr. Stucky's paper. He thought, however, that the benefit which Dr. Stucky's patients derived was twofold—first, from an increased standard of health; and second, he did carry out a certain amount of local treatment, which was the secret of his success. In any aural disease the promotion of the general standard of health influences the aural disease favorably.

Dr. Stucky had spoken of gastrointestinal types of this condition. All have seen this in otitis media cases which have vertigo. Here, treatment directed to the gastrointestinal tract produces good results, while local treatment is not of much value. In other cases of otitis media, however, no such happy results are obtained. The cases suffering from vertigo are not improved if the symptoms are due to disturbance of circulation. Here, again, the treatment must be directed to the cause.

In other otosclerotic types the history shows no trouble with the nutrition or circulation, no gastrointestinal disturbance. The patients deny these, and yet they have otosclerosis. While Dr. Stucky had not spoken of these cases, the otologists owe them attention. There is a general tendency that local treatment is not of much avail. Dr. McKernon said, however, that he believed in these cases of slowly developing otosclerosis, local treatment applied to the tube, ear, nose, or nasopharynx can accomplish a great deal, and perhaps arrest the disease for some time. Especially was this true when general systemic treatment was used in conjunction with the local. A large series of cases coming under his observation during the past few years has tended to confirm him in this opinion. It is difficult to say what is the etiologic factor in otosclerosis. The thanks of the section were certainly due to Dr. Stucky for bringing this subject before the members.

Dr. Dench thought Dr. Stucky's paper very opportune, as the members of the section had been accustomed to hearing so large a proportion of surgical papers. One point especially worthy of notice was that these patients usually come under close observation only after the second or good ear has become more or less seriously involved. We are naturally endowed with so much more hearing than is ordinarily required, that small degrees of impairment indicating the inception of disease go unrecognized. The patient may not even know that there is profound impairment of hearing in one ear, and it is only when the other or good ear becomes much affected that he applies for relief. It would seem to be most desirable that the family physician should test the hearing of his patients at least once a year, to detect the beginning of any aural disease. If such patients came under observation early, diet and other general measures would do more good.

Dr. Dench said that he believed vertigo was a common symptom of otosclerosis. At the present time it seems difficult to know just which cases do have otosclerosis—whether impairment of hearing with normal middle ear and labyrinthine changes constitute it, or whether labyrinthine changes alone are present. If these middle ear conditions do exist, they must, for the most part, involve the oval and round windows.

Dr. Neumann, of Vienna, during a number of years, had found at autopsy only fourteen cases of otosclerosis. This would emphasize the fact that the disease is not so common as is generally supposed.

He wished to confirm and emphasize Dr. McKernon's remarks on the value of local treatment. He has seen improvement occur from local treatment in a large number of cases, as shown by subsequent tests for hearing, including that with tuning forks—the lower tone limit having become lower. This surely shows the value of local treatment. He believes in proper systemic treatment when indicated, but the value of local treatment should not be overlooked. He has made the diagnosis by tuning fork and other tests, and yet treatment has resulted in great improvement. These patients should be treated until it is demonstrated that treatment is of no avail.

Regarding the importance of systemic conditions, the ear is the organ of least resistance in these cases, and therefore becomes involved, and in such cases local treatment combined with proper general treatment should do good. Strenuous living seems to have a great deal to do with the causation of progressive impairment of hearing.

Dr. T. P. Berens thanked Dr. Stucky for calling attention in so able a manner to this much neglected phase of otosclerosis. Many cases of otosclerosis, accepting Dr. Stucky's definition, had been benefited by correction of faulty metabolism. During the past ten years he (Dr. Berens) had referred many cases to internists with marked benefit; particularly in preventing the only too frequent acute exacerbations of the disease. He believes that otosclerosis, as defined by Dr. Stucky, is in a great many ways similar to arteriosclerosis, and that its treatment should be conducted along the same lines as the latter.

Dr. Duel said that one must distinguish between cases of otosclerosis and cases of catarrhal otitis media. In his own experience he had come to regard as cases of otosclerosis only those with definite history of one or more ancestors who, like them, in early adult life (twenty to thirty years of age) had begun to develop progresive deafness. In his experience, local treatment in such cases had usually proved ineffectual. But in the socalled catarrhal type, in which the impairment had been due to some lesion in the conducting mechanism, improvement had usually resulted from vigorous local treatment. We have not yet definitely agreed as to all the causes of otosclerosis, but results forced nearly all to admit that treatment was incidental. He had observed some cases continuously for seventeen years, which had received a great deal of local treatment. He did not think that the local treatment had to any degree influenced the change in hearing which has taken place, though the moral support of such treatment might have been of value. It was difficult to say with any certainty whether or not such cases are influenced for the better by any local treatment.

Dr. Stucky's results with little or no local treatment seemed to be as good as any one could show following vigorous treatment. Dr. Duel then cited the case of a patient whom he had seen two days previously, after a long interval. She had first come under his observation fourteen years before. He had "strenuously" treated her for several years; then two other otologists had treated her for several years; then she had been under the care of a Christian Scientist for several years more—and today her hearing was practically the same as it had been fourteen years ago.

The change in the labyrinthine capsule seemed to go on, despite all efforts to check it; in some cases, rapidly; in some, very slowly; in others, imperceptibly. If we can learn enough to influence otosclerosis by change in mode of life, etc., or find some internal treatment to check its advance, something may be done in generations to eliminate the disease. Perhaps something could be done by eugenics. He firmly believed that it was only through control of marriage of otosclerotics that the disease could ever be blotted out.

Dr. Hays said that one point of importance had not been brought out in the discussion. These patients often apply for relief when they are very deaf and are beyond the treatment of the cause of the condition. Certain connective tissue changes have taken place within the middle ear, similar to those which take place in cirrhosis of the liver and locomotor ataxia. It seemed evident that although much can be accomplished by general treatment, still a great deal of local treatment must be employed in order to overcome some of the connective tissue changes.

Dr. Sheppard wondered what the various gentlemen meant when they spoke of cases of "real otosclerosis." Our German colleagues have shown that Bezold's triad is not necessary for a diagnosis of otosclerosis. A review of the literature preparatory to writing a paper on the subject last spring, made it clear to him that he did not know what it was, and induced him to believe that no one else did. We clearly need more autopsy work. Is it due to disease of the middle or internal ear? Does it consist of Bezold's triad, with a normal drum and middle ear? One German writer on the subject found that a large percentage of his cases did not show Bezold's triad, but rather auditory nerve deafness. He agreed with Dr. Stucky as to the lack of value of local treatment.

If the middle ear, tube, nose, and nasopharynx are normal, why treat them? He has had no considerable number of cases in which relief was brought about by changes in metabolism, as cases with bad metabolism have abnormal mucous membranes, and are therefore not cases of otosclerosis. We should not discourage these patients, but should do our best by building them up to perhaps hold some of them stationary. As to eugenics, he had had two propositions put up to him. First, the family physician had referred to him a young woman who had otosclerosis and was sterile after several years of married life. The doctor was being urged to do some operation to remove her sterility, and the question arose whether it would be advisable, in view of the fact of her having otosclerosis. He advised letting her alone. The second instance was that of a woman who had otosclerosis (her mother also having had it) and who wished to get married. The question arose whether her hearing would get worse, and whether it might be transmitted to possible offspring. He had felt compelled to admit that both of these things might happen.

Dr. Stucky, in closing the discussion, expressed his appreciation of the free and candid discussion of his paper.

Replying to Dr. McKernon, he said that the vertigo spoken of in these cases was undoubtedly due to some circulatory disturbance, and that this disturbance was due to faulty metabolism. This was a point which he wished to emphasize, and which must be corrected. He had frequently seen cases in which the urine tests and microscopic examinations showed nothing wrong and the blood pressure was normal. One case had been examined by the internist and himself carefully at different times, and at no time could they discover any possible cause of the progressive deafness; a few months later the disturbing factor was discovered by a neurologist to be an emotional perversion. After several months of treatment by the neurologist this was relieved, and the patient slowly showed evidence of improvement, in two years the hearing being improved as much as five per cent.

There are periods in all these cases when there is a sudden decrease in the loss of hearing power, and these periods can be likened to the explosive attacks of gout. In many gouty cases there is a normal condition of the urine with slight variance in the blood pressure, and the acute attack of the gout is accompanied by sneezing and all the evidences of acute rhinitis. Both the rhinitis and the gouty pains are speedily relieved as soon as the secretions are hyperalkalized and the alimentary canal is emptied and cleaned.

Dr. Stucky said that he had not intended to convey the impression that no local treatment was ever indicated in these cases, but wished to emphasize the point that he gives no local treatment in the way of inflations, vibration, or electricity unless there are positive indications for the same.

He quite agreed with Dr. Dench, in saying that there may be vertigo in the nonexudative catarrhal otitis media as well as in otosclerosis, and expressed his regret that Dr. Dench had not explained how he accounted for its existence in such conditions. If we exclude all possible localized causes of the vertigo and tinnitus, there being no evidence of middle ear, eustachian tube, or nasopharyngeal diseases, then the cause must be systemic. At any rate, in his own hands the greatest relief of these two conditions had resulted from systemic treatment. Probably no otologist would question the fact that there was danger of overtreatment by inflation and mechanical manipulation.

He quite agreed with what Dr. Duel had said, and had only referred to pregnancy, heredity, and eugenics in the paper as possible causes of otosclerosis. Personally, he had not much faith in the hereditary factor. One might inherit a tendency to this disease, but not the disease itself; and in some cases of this evidence of faulty metabolism in the child born of a parent who had otosclerosis, he believed the cause of the manifestation to be because until adolescence the child lives very much the life lived by the parent, eating the same kind of food, and with environments tending to cause faulty metabolism. He said, further, that if we begin early enough with a child who has an hereditary tendency to this disease—as he had been able to do with four cases within the last twenty years—that he believed they could be prevented from developing the disease. Pregnancy and parturition for the time being lower the vitality, and for the reason that "a chain is no stronger than its weakest link," the ear, if previously weakened, begins to manifest evidence of the disease. In the series of cases presented in the paper, three of them had otosclerosis and had borne children, one having had six children, another four, and the third two. Pregnancy and parturition did not increase the deafness; on the contrary, there had been a slight improvement in hearing.

He expressed his indebtedness to Dr. Duel for the very appropriate phrase, "strenuous treatment," which in his own hands had done more harm than good. The improvement noticed from change of climate, rest and outdoor life was the result of arrest of imperfect metabolism, caused by living a more normal life.

Dr. Kerrison had summarized the discussion so thoroughly that it was needless to add anything to his excellent comments. Doubtless all present agreed with Dr. Sheppard when he said that "We all know so little about the disease that we are in danger of becoming nihilistic." Rather than do that, however, we should not only hold out hope to our patients, but be ourselves filled with the greater hope and determination to ascertain the cause of the disease and devise a mode of life and plan of treatment which would remove it. When we show the same interest and determination in dealing with cases of nonexudative otitis media and otosclerosis that we do with syphilitic and tubercular patients, and are willing to give them the same time, care and perseverance, we will begin to get results. Certainly nothing can be promised in these cases in less than a year's treatment. Each case is a law unto itself. The mode of living of the patient must be regulated as well as the diet. Reconstructive alteratives must be persisted in for a long time. Internal secretions must be regulated by such remedies as will accomplish these. In a word, our efforts must be directed to changing the faulty metabolism which results in these diseases into a normal metabolism which will relieve them. That progress has been made along this line and that a greater progress will be made in the future, there was little doubt. At any rate, in the treatment followed in the cases reported, and in others still under observation, in the long self-denial and rigid observance of certain rules and regulations, the patient has everything to gain and nothing to lose; and of those who are impressed with the deprivation and sacrifice which the future brings with defective hearing, a few are willing to abide by the rigid discipline which in the majority of instances gives such relief and encouragement that they are unwilling to return to the strenuous abnormal method of living.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

Meeting of December 13, 1912.

Paper: Vertigo: A Clinical and Therapeutic Study.*

BY MR. RICHARD LAKE, F. R. C. S.,

LONDON.

DR. KERRISON said that Mr. Lake's name was so closely associated with his pioneer surgical work for the relief of vertigo, that many of his auditors had probably expected that his paper would deal very largely with a discussion of surgical cases. Instead, he had contented himself with a very guarded and conservative statement as to the indications for surgical intervention in these cases, and had occupied his time very largely with a broad and philosophic discussion of the various causes of vertigo.

DISCUSSION.

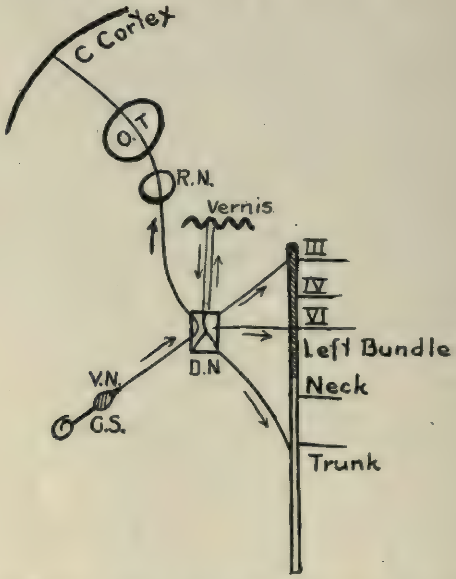
DR. CHARLES L. DANA said that he felt something like an intruder in the discussion, especially after listening to Mr. Lake's admirably presented paper, which contained so many points worthy of discussion and remark, and which he personally felt unable to criticise. He expressed his appreciation of the invitation to join in discussion of the subject, and was the more inclined to accept it because of his own rather definite views in regard to vertigo. He was interested to know whether these views were sound and would stand criticism.

It has been his belief that all vertigo is a vestibular affair. Vertigo is not always aural, but it always has to do with a disturbance in the function of the vestibular mechanism. Vertigo is a kind of neuralgia or paresthesia of the eighth nerve. The vestibular part of this nerve with its central tracts and connections make a very complex mechanism. The vestibular fibers originate in the ganglion of Scarpa and sup-

---

***See page 970, December, 1912, number of Annals.**

ply the semicircular canals; then the nerve goes to the three vestibular nuclei in the medulla, which form its most important central station and a primary vestibular center; from there these nuclei send out axites in several directions. Some pass through the longitudinal bundle to the nuclei of the oculomotor nerves. Some few fibers pass up to the vermis of the cerebellum, but the connection here is very slight. The cerebellum is not the organ of the space sense, as was once thought. The vestibular nuclei send afferent fibers to the red nucleus, and thence on to the optic thalamus, thence to the cortex of the brain. We do not know that the anatomic and physiologic relations cease there, for there are tracts from the cortex passing down to the pons and cerebellum, forming thus a circuit; so that in order to keep ourselves straight in the world, we have to play upon a very important and elaborate mechanism. There is not a moment in our lives when we are



The vestibular nerve and its connections. The mechanism of equilibrium tonus is the V. N., vestibular nerve. D. N.—Deiter's and other primary nuclei. Nucleus in vermis. L. B.—Longitudinal bundle and its connections with oculomotor nerves and anterior horns. The sensations of vertigo are sent up through the red nucleus and optic thalamus to the cortex.

not doing something to keep ourselves properly balanced in space.

The vestibular tracts are all afferent; they form a sensory nerve which carries impulses into the brain. When this is irritated, it causes a disturbance of sensation which is analogous to a pain or dysethesia, and which we call vertigo. This sensation is to the vestibular nerve and its central tracts and stations what pain is to the trigeminal nerve, and that is the basis on which we can explain the phenomena of vertigo. Just as the trigeminal nerve can be stimulated and pain be aroused by irritation of the peripheral part or of the roots, of the Gasserian ganglion, or of the central tracts, so the vestibular nerve can be irritated in the ear and labyrinth, in the vestibular root, the three vestibular ganglia and in its more central parts, thus arousing sensations of vertigo.

Dr. Dana said that while there is a large number of cases of aural vertigo, there is a much larger number of cases of vestibular vertigo, not at all aural. In neurologic and general medical practice we see ten cases of severe vertigo in which the ear is not the slightest degree involved, to one case in which the trouble is distinctly aural—at least that was his experience.

Most of the nonaural cases of vertigo can be explained on the theory of disturbance of the more central part of the vestibular mechanism. There is a disease known as migraine, which is a periodical sensory neurosis due to discharges from the cerebral cortex.

Sometimes instead of having migraine, a person will have an attack of vertigo. The speaker said that he had had a number of patients with attacks of this kind of migraine—vertiginous attacks—with Meniere's syndrome taking the place of the headache; other cases had vertiginous attacks lasting several hours, followed by severe pain and the sickness of migraine; in other words, the vertigo was part of the aura of migraine, just as zigzag lights are part of the aura. That would suggest that some forms of vertigo are cortical and due to cortical discharges. There is also epileptic vertigo which is known to be cortical.

There are some organic nervous diseases in which vertigo is one of the common characteristic symptoms; one of these is multiple sclerosis. In this disease the pons and medulla are very often affected, perhaps more so than any other part of the central nervous system; and here the central paths of the vestibular nerve are affected. This theory of vertigo has been offered in cases that came to his clinic, and he has much simplified the explanation of the phenomena.

In regard to some of the forms of vertigo which are not aural, but which are very striking in their characteristics and have very distinct clinical syndromes, one form appears to be like a discharge from the cerebellar cortex. The cerebellum is not a center of the vestibular nerve, but the cerebellar nuclei do send fibers down to the vestibular nuclei, and the cerebellum is thus in close connection with the nuclei of the vestibular nerve; therefore it seems a reasonable theory that in these attacks where there is cerebellar disease and vertiginous attacks, the latter are due to the action of the cerebellar discharge upon the vestibular nuclei.

There are some interesting clinical facts regarding vertigo which are of special value to the neurologist. There is a condition which is sometimes aural, more often not, in which the patient does not have vertigo, but has the sensation that he is going to have it; a sense of insecure equilibrium, a sense of uncertain balance so great that the patient calls a cab and goes home for fear of falling in the street. Analysis shows these cases to be psychical in character; so that we have a vertigo of a type which may be called psychic, or, if you wish, hysterical.

Another form of vertigo which may have something to do with aural conditions is chronic or permanent vertigo. A patient of the age of twenty or thirty becomes dizzy and continues so for years during waking hours; this is not an objective vertigo, but a subjective one. The patient may go about attending to his duties, or may even play lawn tennis, but has always a certain degree of dizziness. That is known as permanent vertigo. This form sometimes runs through families. It has been described by Oppenheim.

The doctor said that he had listened to Mr. Lake's paper with great interest, and would like to discuss it at length. One point had much impressed him. Recently he had been told of a case of aural vertigo of a severe type in which it appeared there had been a hemorrhage into the labyrinth. The patient got well. There was no history, so far as was known, of syphilis, but two and a half years later the man had a general paresis. In his own experience, quite a large percentage of cases of aural vertigo had been of specific origin. Mr. Lake had included paresis in his list, but had put very slight emphasis upon it, whereas it would seem to be a very important cause.

Dr. McKernon said that he had nothing to add to the paper, and only wished to express his hearty approval of it. The classification which Mr. Lake had made was admirable, for several reasons. In the first place, it was simple, and in the second, it could be easily applied; the cases could easily be grouped and followed.

Then he had been forcibly struck by the very small number of operations which Mr. Lake had found necessary to perform in the number of cases of distinct labyrinthine disease which he saw. This was very significant. Since taking up this work on the labyrinth and studying it more closely, he has come to the belief that at the present time—as well as in the past—there is too much indiscriminate exploration of the labyrinth; the labyrinth is opened too frequently, without definite and positive indications.

One very interesting point which Mr. Lake had brought out was the necessity for taking the blood pressure. He had also made another important point, i. e., that vertigo is a symptom and not a disease, and that its relation to other systemic conditions must be taken into consideration. He had also spoken of the advisability of not operating in cases where the hearing was good. This was in line with the views held in this country at the present day.

It was very interesting to notice the association of the low blood pressure with the hypersensitive vestibular apparatus. That emphasizes to a much greater extent than we have probably taken into consideration, the advisability of taking the blood pressure in all cases of labyrinthine disease when they first apply for examination.

Dr. McKernon said that among the several causes which Mr. Lake had mentioned, he had not heard him refer to exostosis of the external auditory canal. He himself knew of several cases in which these had been present and had caused vertigo. He also mentioned several cases coming under his observation of men addicted to the use of alcohol to a great extent. He had seen several such cases accompanied with labyrinthine hemorrhage and followed by complete and permanent loss of hearing.

Mr. Lake had also spoken of the lack of danger in doing the operation upon the labyrinth. He was certainly to be congratulated upon the results reported, and in his hands the operation may be devoid of danger, but in operating upon the labyrinth with either suppurative or nonsuppurative conditions, it has to be done with a view of danger in the future from complications arising.

Mr. Lake had also spoken of pilocarpin being given for two weeks. In this country it has been the habit to give it over a prolonged period of time, usually six weeks. He had also spoken of the first symptoms of arteriosclerosis in certain cases being vertigo. This was a very important point, and should lead to a more thorough investigation of these cases from a systemic standpoint than we have been giving them.

In speaking of the drugs used for this disease, Mr. Lake had not mentioned one which in Dr. McKernon's experience with cases of vertigo and high blood pressure had proved very valuable, namely, glonoin.

The members of the profession in this country were certainly very fortunate in having Mr. Lake come to us and give us the result of his wide experience in labyrinthine work.

Dr. Dench said that he was greatly indebted to Mr. Lake for the classification of the symptom of aural vertigo. He believed this classification was the best that had ever been presented to the medical profession. Dr. Dench remarked that he recalled several cases of aural vertigo which had come under his observation about which he would like to speak. In three of these a middle ear lesion was present. One of these patients had been previously examined by a prominent otologist, who had pronounced the case not one of aural vertigo. In all of these cases the vertiginous symptoms were extremely well marked. In two of the patients the attacks of dizziness were so severe as to prevent them from attending to their regular work. One of the patients, when first seen, had such frequent and severe vertiginous attacks, that it was necessary for him to go about with an attendant. In all of these cases the introduction of two drops of a 2 per cent solution of pilocarpin into the tympanic cavity, through the eustachian catheter, was followed by a complete disappearance of the vertiginous attacks, and by great improvement in the hearing. One of these patients had been under occasional observation for over a year and a half, and had had no vertigo for over one year. All three patients were now attending to their regular occupations without inconvenience.

In another class of cases operations upon the middle ear were indicated and were of distinct value. In one case the malleus, incus and drum membrane had been removed and the oval window opened, although the stapes could not be extracted. This patient had had no vertigo since the operation. The hearing, however, was at present completely lost.

Referring to the suggestion of Mr. Lake, that increased blood pressure may act as a causative factor in these cases, Dr. Dench believed that two factors were always present in disturbed blood pressure in cases of aural vertigo. One was a previous lesion, either within the tympanic cavity or within the labyrinth, and the second was a disturbance of the blood pressure. The lesion of the internal or middle ear would be reevaled by tests of the hearing and of the static labyrinth. Where the blood pressure factor and the aural factors were combined, the relief of either factor would be followed by a cessation of the vertigo. For instance, if the blood pressure is increased, and the aural lesion is slight, such drugs as glonoin or the iodids, which will reduce the blood pressure, might be sufficient to overcome the vertiginous attacks without any aural treatment; or, if the blood pressure were not too far removed from the normal standard, the relief of the aural condition, provided this were in the middle ear, might also relieve the vertiginous attacks, and the blood pressure factor of the vertigo might be disregarded. The same could be said of those cases which are supposed to follow intestinal toxemia. Here Dr. Dench believed that there was always some lesion of the auditory apparatus present which determined the character of the symptoms. The labyrinth being in an unstable condition, as it were, due either to some organic change in the labyrinth itself, or to some disturbance of the labyrinth secondary to some pathologic change in the middle ear, was more easily affected by a toxin than it would be under normal conditions. Hence, the patient had an attack of vertigo as the result of his intestinal toxemia, rather than some other manifestations of the toxic condition.

With reference to specific cases, Dr. Dench reported the case of a patient who was suffering from a very mild middle ear lesion, involving both ears. This patient had severe attacks of vertigo. The Wassermann reaction was positive, bone conduction was diminished, and the galvanic test showed hyperexcitability of the auditory nerve. This patient had been under antispecific treatment, but the symptoms had not been relieved. He was finally given heroic doses of mercury, and made a complete recovery.

Dr. Dench also called attention to the value of taking the galvanic reactions of the auditory nerve in every case of vertigo, and he believed that a great deal would be learned from a careful study of these reactions.

The speaker said also that he wanted to confirm what Dr. McKernon had noted regarding the internal administration of pilocarpin. No effect could be expected from this drug unless it was administered regularly for a period of from six to eight weeks.

Dr. Philip Hammond, Boston: I am sure we are all indebted to Mr. Lake for the admirable classification of labyrinthine diseases which he has presented this evening. A paper such as we have just listened to is epoch making, in that it deals with a form of vertigo which is but little considered. Much is appearing at the present time in our medical publications concerning the vertigo of labyrinthine irritation, particularly in suppurative cases, but little progress has been made in the study of this affliction in nonsuppurative otitis media. The mere mention of the term aural vertigo generally calls to mind Meniere's disease, and for the average man this is an all comprehensive term. We all know about the original case described by Meniere, and although this name has been fastened on to thousands of similar attacks since then, there have been but about fifteen authentic cases verified at autopsy. Because there are so few cases of vertigo which can properly be called Meniere's disease, and because there are so many patients made dizzy by apoplectiform seizures which in some respects simulate the classical description of this disease, many writers believe with Amberg that it would be better to drop the name Meniere's disease from aural nomenclature, adopting in its stead some more definite term. Therefore, we should be doubly grateful to Mr. Lake for presenting for our consideration examples of other pathologic conditions which are capable of causing vertigo.

As has been intimated in his excellent paper, which we have just heard, progress in our knowledge of this condition is slow, particularly as we have so few cases that can be examined post-mortem with any definite history of the condition while living. It is necessary that we group together the known facts relative to this disease before any attempts at theorizing are possible. It is only within recent years that a discrimination has been made between true Meniere's disease and aural vertigo of apoplectiform type, and it is only recently that increased labyrinthine pressure has been thought to be the cause of such attacks.

I am very glad that Mr. Lake has expressed such decided views on the question of increased pressure as the cause of vertigo. He tells us this evening that in none of the cases on which he has operated has he seen fluid under pressure in the labyrinth. Now this finding has been confirmed by the experience of one of my colleagues in a case which he reported before the International Otological Congress in Boston last summer. I refer to the case operated upon by Dr. Walker. In this case there was some fluid in the labyrinth, but the quantity was so small that it did not even run out after opening the bony capsule. Now this at once leads us to believe that some definite change has taken place in that part of the ear which forms the perilymph, otherwise there would have been in these cases the escape of a quantity of fluid.

That the perilymph must be able to run into the labyrinth freely from some source is shown by the quantities which are secreted following the evulsion of the stapes or accidental opening of the bony labyrinth. So good authorities as Schwalbe and Siebenmann assert that there is no definite connection between the labyrinth and the subarachnoid space. What seems to be highly probable is that there exist small passages, as the aqueductus cochleæ or spaces around the internal auditory meatus, or both, through which fluid may percolate. Because of the conditions found in these cases reported by Mr. Lake, and confirmed by the observations of Walker, it would seem highly probable that in certain cases of otosclerosis these natural fissures through which fluid comes to the labyrinth are more or less, if not absolutely, obstructed. While increased pressure may not be the direct cause of the vertigo, it has been demonstrated that sudden changes in pressure do cause it. It has been observed that in practically all cases of aural vertigo there is a certain amount of deafness gradually increasing, due either to otosclerosis or to an adhesive process around the stapes and round window, thereby eliminating one of the normal means for allowing for variations of pressure in the labyrinth.

In order to seek for a means of curing this disease, one must look for the cause of the suddenness of the attacks. As has been shown by Mr. Lake, many of these attacks come on while the patient is absolutely quiet, and in fact in some instances during sleep. In these cases it is very difficult to imagine that the vertigo is caused by a gradual increase in the labyrinthine pressure, but it is very easy to imagine that it is the result of a sudden lessening of that pressure. For instance, in glaucoma there is ample opportunity for expansion of the eye with the increase in pressure of the fluid within it. In the ear, however, we have no such opportunity for expansion and increase in the quantity of the fluid, and there is very definite and very severe pressure brought to bear upon all parts of this delicate mechanism. Now, assuming that we have such an increase in pressure within the labyrinth, one can imagine that after reaching a certain limit something must give way. The labyrinth, as is well known, is surrounded by a bony capsule having no definite apertures leading therefrom.

The first decided step forward in the treatment of aural vertigo was the introduction of lumbar puncture by Babinsky. Molard agrees with Babinsky in his conclusions that great relief is attained in cases of vertigo following this procedure, the beneficial effects lasting in some cases more than a year. They both state that the symptoms are frequently augumented immediately following the puncture, improvement coming only after a period varying from several days to a week. In his investigations Babinsky ascertained first that a galvanic current passed through the head causes vertigo in normal human beings; second, that with otosclerosis or vertigo there was a greater resistance to the current; and third, that this resistance was reduced following lumbar puncture. Reasoning that the strength of current required to cause vertigo when passed through the vestibule was diminished following lumbar puncture, he thought that without doubt the lessening of the pressure in the spinal canal affected the pressure in the labyrinth.

His conclusions, based on the study of a considerable number of cases, were that vertigo is almost always lessened, that the hearing might not be improved, and that the distressing tinnitus persisted. This work of Babinsky has been more recently confirmed by the investigations of Blake and Putnam in Boston.

Experiments through many years, beginning with the work of Ewald on birds and animals, have shown that the sense of equilibrium is situated in the semicircular canals and vestibule. These studies have been confirmed by the more recent work of Neumann, Bárány and others on the infected labyrinth in the human body. Our most recent knowledge leads us to believe that motion imparted to the hair cells on the crista ampullaris by the endolymph as it moves in the semicircular canals, causes sensations of change in position, and if the vestibule with its ampullæ be destroyed, the ability to perceive changes in position will be lost. In other words, the vestibule is the important thing to destroy, and the semicircular canals are only of secondary importance.

We are indebted to our English colleagues, Milligan and Lake for our knowledge of the most radical procedure in these cases of nonsuppurative labyrinthitis. While this work is intensely interesting and instructive, and it is hoped that much good may come from it, it is certainly not a method that will be employed for the relief of all cases of vertigo, nor is it advocated as such by its originators.

This brings us to a consideration of the means at our command for combating this malady. First and foremost is some method of depletion. This we are accustomed to accomplish in many ways. Pilocarpin subcutaneously, the use of a seton, the administering of large doses of salts, and the ingestion of adrenal extract, as suggested by Randall, are among those in use. If these fail to relieve, the advisability of lumbar puncture should certainly be considered.

Finally, should we resort to this radical operation on the labyrinth, the subsequent history should be most carefully followed, as we must remember that almost equally good results are attendant upon lumbar puncture—but they are not permanent. Opening the labyrinth would be but a more direct way of reducing intralabyrinthine pressure, were we to stop at the simple opening. The whole subject is one of most absorbing interest, and promises to open up new fields for endeavor in our special work.

Dr. Percy Fridenberg: We have to congratulate ourselves and to thank Mr. Lake for a careful, practical study of an abstruse symptom complex, vertigo, from the clinical point of view, which, as he has pointed out, gives us the firm basis of observed fact from which to start on our deductions, and which is the only really promising one, at least in our present knowledge, or lack of it, regarding the pathology and much of the physiology of equilibration. We have become accustomed to accepting without question certain conclusions which are but little more than inferences. This applies, for instance, to the etiologic factor, so freely accused, of arteriosclerosis. We know that arteriosclerotics often have vertigo, or rather get dizzy, but so do the neurasthenic and the hysterical. What is the underlying structural or functional change? If it is anemia secondary to narrowing of arteries and slowed blood current, why is the vertigo not continuous? Why do we not get severe vertigo in other blood states characterized by insufficient circulation or nourishment? There is no doubt that circulatory disturbances play an important role in the causation of vertigo, and that hyperemia and congestion, acute or chronic, may be as deleterious as the opposite condition. I need merely refer to the vertiginous states induced by nitroglycerin and similar vasodilators, the vertigo of acute alcohol intoxication, and the dizziness produced in plethoric subjects by sudden or prolonged stooping. That circulatory conditions, too, are of clinical importance in true labyrinthine vertigo, without actual suppuration and pressure from exudate, seems to me at least probable. Hyperemia of the internal ear or serous labyrinthitis must be considered. I believe that we may even have a transient condition of hyperesthesia of the labyrinth without morbid changes, due to toxins or transitory irritation or vascular circulatory anomalies, and this I should like to call labyrinthism rather than labyrinthitis, just as we speak of meningism. I doubt whether an actual pathologic contraction of either the semicircular canals or of the other tubes mentioned by the reader could be held responsible for a transient or intermittent vertigo. It could of course explain a continuous symptom of this sort, but it would be difficult to understand how the vertigo could be relieved spontaneously as long as the narrowing, if that really was the cause, remained, as it would have to do on a permanent histologic basis. Among the factors of a reflex labyrinthism or of a vertiginous state we must consider eye strain, especially the variety dependent on imbalance of the ocular muscles. When there is actual diplopia, as in ocular palsy, the vertigo may be so extreme as to cause nausea and vomiting and require the exclusion of the affected eye from the visual act.

In the individual case we may have to decide whether the coexistence of deafness with vertigo or of specific disease with vertigo indicates a casual relation or merely a fortuitous coincidence, and this question is by no means academic. Considering our limited knowledge of the pathology and minute physiology of the inner ear, we may only be able to arrive at a decision ex juvantibus.

In connection with specific disease, I wish to refer to salvarsan injections, and to the fact that attacks of vertigo with other marked systemic disturbance are not rare. Various theories have been advanced, but we are still in doubt whether we have to deal with acute toxic (arsenical) vestibular neuritis, labyrinthine hyperemia due to setting free of a certain number of spirochetæ in the blood stream, or a Herxheimer's reaction.

The arbitrary age limit of forty by no means excludes arteriosclerosis, at least in America. This fact might explain the variable result of operative treatment and the necessity of making two subdivisions of classes, those benefited, and those unimproved. The latter were possibly juvenile arteriosclerotics, or at least premature presenile arteriosclerotics. We may in time to come realize that there are other changes, possibly in nerves and end organs, as well as in bony tissues and structures, quite analogous to arteriosclerosis in their deleterious effect on function, and that such morbid processes, like certain low grade intoxications and infections, may well produce a gradual and slowly progressive deafness, together with nutritional and circulatory disturbances, giving rise to vertigo and vertiginous states. Gastrointestinal or renal autointoxication may produce a symptom complex closely simulating brain tumor, with nausea and vomiting in the foreground of the clinical picture. How much of this is due to toxic causes and how much to increased intracranial pressure? We know, since Cushing has pointed it out to us, that the choked disc of

Bright's neuroretinitis is not due only or even mainly to toxic and vascular anomalies, as was accepted traditionally, but is a manifestation of high pressure within the skull. Similar factors acting in less intense degrees would undoubtedly produce vertigo.

As to labyrinthine hemorrhage, it seems probable that this may occur at a comparatively early stage in life. While it is true that the hemorrhages due to pronounced atheroma are characteristic of senility, we may, I believe, have presenile changes, just as we have them in the retina, where, too, as I may remind you, it was formerly thought that hemorrhages were only found in old age. Various blood states, leukemia, malaria, pernicious anemia, purpura, septic systemic and local infections, and finally some toxic drugs and mineral poisons, predispose to, and excite, capillary hemorrhages in comparatively young subjects.

Tinnitus might be explained as a symptom of autogenous irritation, depending on paralytic dilatation of blood vessels in the cochlea following complete obstruction, as in partial thrombosis or transient spasm. This is quite analogous, clinically and pathologically, to visual symptoms of flashes of light observed in cases of vascular spasm of the retina and in the dazzling scotoma of hemicrania, which is undoubtedly due to localized brain anemia.

Among traumatic agents we must consider the effect of sudden and intense stimulation by sound. It would seem not at all improbable that very high pitched or penetrating tones might cause such vibration as to tear a number of delicate vessels in cochlea or labyrinth. I have known the low pitched vibrating hum of a locomotive exhaust to produce most annoying symptoms of confusion very much like vertigo.

Persistence of equilibrational disturbances, as inability to walk in the dark, after the cessation of tinnitis, would seem to indicate that neuritis of the vestibular nerve had progressed to degeneration and complete loss of function, which was then to be compensated, in time, by the contralateral labyrinth.

Repeated and sustained emotional excitement may produce effects not unlike those of arteriosclerosis, of which it is again a frequent cause. Vertigo in the morning is not infrequently observed as a result of sexual erethism, continued and unrelieved by the natural climax and consequent relief of tension, physical, vascular, and emotional, and in such other emotional stress as continued apprehension of loss of money, or of reputation, worry, anger.

We must bear in mind the possible influence of toxic factors, especially in gouty subjects and in some of the cases with diminished blood pressure.

Vertigo with ocular symptoms borders on the migraine complex, which, as we know to our regret, is not at all uncommon. The account of the case of visual aura followed by intense vertigo and nausea certainly suggests eye strain with scintillating scotoma. Vertigo coming on with bright light, as reported, or, and this is curious, with loud noises, suggests retinocerebral vascular spasm, or the peculiar synergy known as photophonism or phonophotism, if you prefer, noted in not a few subjects of unstable emotional equilibrium. With these subjects every perception of sound produces a sensation of color, and vice versa; they "hear" red, green, blue, as the case may be, with the tones of different instruments; the violin is green, the clarinet yellow, and so on.

A visual or rather an ocular component may be present in a number of forms of what we may term reflex vertigo. Thus in the vertigo of seasickness, it is hard to say just how much is due to the constant changes in position of surrounding objects, how much to splanchnic irritation, how much to actual rotation involving not only the semicircular canals but the entire brain and meninges. The effect of constrained eye positions and abnormal muscle action, intensified by eye strain and retinal torpor due to insufficient illumination, is seen in miner's nystagmus, which is often associated with what must be a purely reflex vertigo. Even lesser degrees of muscular imblance may, as has already been stated, cause similar vertiginous states. It imposes on us the responsibility of having a careful and complete ophthalmoscopic examination made of such cases as are not evidently aural, and even in these we may, as I have done, make the interesting and rather surprising discovery of latent and unsuspected eye strain.

Dr. T. P. Berens said that he only wished to emphasize what had been said about the danger of operations on the labyrinth, and to thank Mr. Lake for coming over and presenting this paper, for the excellence of the classification of these cases, and for the unusual clearness with which the subject had been treated.

Mr. Lake, closing the discussion, alluded first to the very clear and interesting remarks made by Dr. Dana, especially upon the influence of the cerebellum upon vertigo.

Dealing then with the criticisms which had been made, as a whole. The first point was the brief allusion he had made to specific disease—lues. This was chiefly because he had omitted many parts of his original paper on account of its length, and specific disease was one. In considering the value of salvarsan and its dangers, Mr. Lake took the opportunity of expressing his astonishment that so powerful a drug should be given intravenously with no precautions and in a standard dose—not considering the body weight, nor the state of the blood, nor its coagulability. As to the intratympanic use of pilocarpin, Mr. Dench was much to be congratulated upon his results.

With regard again to the galvanic tests and reactions in cases of aural vertigo, Mr. Lake said that time had been wanting, and he thought would always be wanting, to allow of all these tests being employed. For time represents the doctor's capital.

Mr. Lake had never regarded Babinsky's work as quite reliable from an aurist's point of view, the absence of tests and proper aural treatment rendering them useless. At all events, the effect of lumbar puncture was, inter alia, to lower the blood pressure, an effect to be obtained otherwise.

With regard to increased labyrinthine pressure, personally he was skeptical as to its existence.

In respect to operation, he had removed all the semicircular canals once, but the shock was very severe and so he proceeded to attack the vestibule. As for the operation being dangerous or not, he believed that turned, as he has hinted, upon the question of strict antisepsis.

In conclusion, Mr. Lake expressed his great appreciation of the honor done him, not alone by the invitation he had received from their chairman, but by the attendance of so many of his colleagues, especially to those who had covered long distances for so short a time.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of November 19, 1912.

DR. JOSEPH C. BECK, PRESIDENT.

PRESENTATION OF CASES.

Sarcoma of the Tonsil or Tonsillar Manifestation of Hodgkins' Disease.

DR. G. A. TORRISON: E. S., 68 years old, engineer, had been troubled for the past week with difficult and painful deglutition. He had entered the Presbyterian Hospital a year ago, where a diagnosis of Hodgkins' disease had been made. Family history was negative. He denied having had syphilis. Inspection of the throat showed an enormously enlarged left tonsil, which reached nearly over to its fellow on the opposite side. The latter was normal in size. There were enlarged glands in the left side of the neck. The surface of the enlarged tonsil was smooth and presented a reddened and glazed appearance, suggesting a large tonsillar abscess. An incision gave no pus, the knife entering a cavity which proved to be an enlarged crypt. The probable diagnosis then lay between sarcoma and a tonsillar manifestation of Hodgkins' disease, the latter being the more likely.

Perforation of Shrapnell's Membrane.

DR. J. HOLINGER: The patient was a painter, 32 years of age. He has been suffering from acute otitis media for the last five days, with very copious discharge. There was a tit-shaped perforation in the region of Shrapnell's membrane. The perforation of Shrapnell's membrane is always due to a chronic process, says Bezold. Politzer, on the other hand, insists that he saw perforations in this part in acute inflammations. This case seemed to be an argument in favor of Politzer's theory. Dr. Holinger slit the tit, and today, two weeks after the demonstration, the tit had disappeared and the perforation was in the posterior upper quadrant. The swelling and the pus must have moved the soft parts. So the case supports Bezold's contention.

Tuberculosis of the Nasal Cavities.

DR. OTIS H. MACLAY presented a case of suspected tubercular destruction of the nasal cavities, the turbinate tissues entirely gone, much crusting, some odor, bony surfaces exposed in various regions of the nose. Antisyphilitic treatment had been tried without apparent benefit; for the last two months has been under injections of tuberculin, with decided improvement.

Paper: Resonators, With Special Reference to the Schaefer Apparatus.*

DR. ROBERT SONNENSCHEIN: Resonators are appliances which have the property of selecting out and reinforcing certain sounds. Resonance is the reinforcement or intensification of sounds due to the union of direct and reflected waves. Resonators have been used in various ways by different men, such as Helmholtz, Schaefer, Abraham, Wien, Wastzmann, and others, for the investigation of many physical problems, among them the analysis of tones, especially the vowels, the study of "interference tones," etc. Although very simple apparatus, such as a lamp chimney, a tube of cardboard, or a bottle, may serve as a resonator, still the best thing is either a spherical resonator or a metal cylindrical tube, both of which may be connected with the ear. If open at both ends, the pitch of a resonator is higher than if one end is closed. On the other hand, a resonator is of deeper pitch (a) the larger the air containing space, and (b) the narrower the sound opening happens to be. Tones of medium pitch are the ones most intensely reinforced, the very low and the very high ones much less so.

The Schaefer set of resonators consists of four cylindrical brass tubes of different lengths, but the same diameter. One end is open, the other closed by a plate having a central aperture. Over the latter is a conical extension intended to be placed in the meatus auditorius, or to be connected with tubing for introduction into the ear. For occluding the small opening when the resonators are not to be inserted into the meatus, rubber plugs are provided. Each resonator tube fits accurately into another cylinder, but can be moved in and out. The inner tube is graduated in millimeters, and at the same time the tones for which the resonator acts at different lengths are indicated by letters giving the notes of the scale. The gradation corresponds to the tones of the temperate scale based on a^1 (435 v.d.), at a temperature of 18 degrees C. (about 64.4 degrees F.).

The advantage of the Schaefer resonators seems to lie in their being compact, accurate, comparatively cheap, and easily adjustable to any desired tone between A (108 v. d.) and c^3 (1024 v.d.).

CONCLUSIONS.

Resonators can be used for various purposes: (1) In physical research, in the analysis of tones, etc.; (2) in testing tuning forks to see if the actual tone is the one claimed to be present; (3) for determining the pitch of unmarked forks or that of any other sounding body whose tone comes within the range of the resonators employed; (4) and last, but not least, for testing the absolute duration of hearing for any tone or set of sounds.

DISCUSSION.

Dr. Richard H. Brown asked whether, if you get a resonator tuned to about a medium pitch, such as a man talking in a very monotonous voice, that can be used by a person with diminished hearing, to catch enough of the sound vibrations to enable that person to hear better? He wondered if that had been tried.

Dr. J. Holinger did not believe that resonators have been used or tried for the purpose spoken of by Dr. Brown, and he did not believe that it would be feasible. The bulk of the resonators excluded their being carried around by a person. One important application, however, might be this: The resonators of the well known span from c^1 to g^1 might be used in determining the degree of defect in hearing in children, in order to find out which deafmutes could be educated through their sense of hearing. If hearing extended over this well known span and amounted to hearing the tuning forks of this pitch for ten per cent of their normal duration, hearing could be used for educating children.

Dr. Edwin Pynchon asked Dr. Sonnenschein if he had had any experience with the audiometer. This instrument had a little bell, the tone of which could be modified as desired by the examiner.

Dr. Sonnenschein, in closing the discussion, added a little to what Dr. Holinger had said in answering Dr. Brown's question. It would not be possible to use these resonators very well as an aid to hearing, for the reason that, while it is true that the range not only covers the range given by Bezold, that is, from b^1 to g^2 (these go considerably lower than that—an octave, and also a full octave higher), we must remember that it is the overtones in the various vowels that give the quality to the voice. Now, even though people speak in a socalled monotone, every vowel has certain overtones, and each resonator will resound only to one particular tone when adjusted at a certain length.

So far as teaching deafmutes was concerned, which was referred to by Dr. Holinger, of course that is very important, but he thinks that the same remarks would apply, namely, that these resonators not only cover this area, but more than that. Regarding Dr. Pynchon's question about the audiometer, Dr. Sonnenschein has had no practical experience with it. It is an apparatus not for reinforcing sound, but for testing hearing by means of a hammer enclosed in a sounding box, which is controlled entirely from the outside, so that the patient has no way of judging what intensity of sound is being produced. This is connected with the ear by tubing as in a stethoscope. One can produce an intensity of sound from 0 to 10, and thus see when the patient begins to appreciate that sound.

Dr. Brown asked whether it is known what the actual difference of pitch is in the vowel tones in monotone talk.

Dr. Sonnenschein replied that even in a monotone the quality of speech depends entirely on the articulation and emphasis, whereas the specific tone would have to have the same pitch. Even if a person speaks in a monotone and pronounces different vowels, they are of different pitch.

Dr. Brown asked what is the difference.

Dr. Sonnenschein said he has forgotten the exact difference, but believes that Wolf showed that there is a range of almost three octaves in pitch of the various vowels and consonants.

Paper: The Physiology of the Semicircular Canals—A Lantern Demonstration.

By George E. Shambaugh, M. D.,

Chicago.

Dr. Shambaugh pointed out the importance of obtaining a correct idea of the anatomy of the end organ in the semicircular canal, and the difficulties encountered in making preparations of these end organs. The end organ in the semicircular canals is the crista ampullaris, which forms a ridge placed so as to receive the full impact of the endolymph passing from the utricle into the canal or from the canal into the utricle. Both sides and the crest of the crista are covered by hair cells. Superimposed above the crista is placed the cupola, which is separated by an appreciable space from the free surface of the epithelium. The hairs of the hair cells project into the under surface of the cupola. The cupola is a stationary cap which is not capable of being displaced by endolymph currents.

ANALYSIS OF THE PHYSICAL REACTIONS WHICH PRODUCE A STIMULATION OF THE HAIR CELLS OF THE CRISTA AMPULLARIS.

1. The stimulation of the hair cells is the result of an irritation applied to the projecting hairs.
2. The irritation of the hairs is brought about by an interaction between the cupola and the hairs.
3. This interaction is occasioned normally by the impaction of endolymph currents against the sides of the cupola.
4. Only those hair cells on the side of the crista receiving the impact are stimulated by a current of endolymph.

Conclusions that are still unsettled in the analysis of these physical reactions are:

1. Is the duration of the stimulation of the hair cells the same as the duration of the endolymph current?
2. Is the duration of the nystagmus the same as the duration of the endolymph?

Since we are able to observe objectively the duration of nystagmus, the answer to these questions can be approached by finding some way of estimating the duration of an endolymph current. Three methods are known by which the duration of the endolymph current can be estimated: (1) The Ewald experiment; (2) the compression test in fistula cases, and (3) the fact that in caloric stimulation of the semicircular canal it is necessary, in order to get an endolymph current, that the canal be placed in a vertical position. The endolymph current which is occasioned in this way by caloric stimulation ceases the moment the canal is placed in the horizontal plane. In all three of these experiments we observe that the duration of the nystagmus produced by an endolymph current stops apparently the moment the endolymph current ceases.

PHENOMENA IN ROTATION EXPERIMENT.

On starting rotation a patient develops a nystagmus in the direction of the rotation. After rotation has continued for some moments this nystagmus disappears. If the speed of rotation is now accelerated, this nystagmus will again return. On suddenly stopping rotation a nystagmus develops in the opposite direction, which also lasts for a number of seconds before subsiding. The simplest explanation for these phenomena was offered by Breuer and Crum Brown, who explained them as the result of inertia of the endolymph. Breuer gave up the idea later, because of the difficulty in accounting for the inertia of endolymph in the small canals producing an endolymph current that would last long enough to explain the duration of the turning and after-turning nystagmus. Breuer accepted the conclusions that both on starting and on stopping turning there can be but a momentary impulse to the endolymph. He still believed that the duration of the nystagmus depended upon the duration of the stimulation of the hair cells. In order, therefore, to account for the stimulation of the hair cells lasting long enough to explain the duration of nystagmus in the rotation tests, Breuer assumes that the momentary impaction of endolymph displaces the cupola. This displacement of the cupola produces a stimulation of the hair cells that continues until the cupola has been drawn back to its normal position. Bárány's explanation for the rotation phenomena is that there are two nystagmus centers, each containing stored energy which requires just so much time to become expended after stimulation. One center produces nystagmus toward the right, the other toward the left. Bárány believes that there is but a momentary impulse to the endolymph on starting and on stopping rotation. This produces a momentary stimulation of the hair cells. The impulse on starting rotation stimulates the center which produces nystagmus toward one side, and the impulse on stopping rotation produces stimulation of the center which directs nystagmus toward the other side.

Fundamental objections to both the Breuer and Bárány hypothesis are the experiments cited above, where in the fistula test and in the caloric stimulation the nystagmus stops the moment the endolymph current stops. If either the Breuer or the Bárány hypothesis were correct, the first impact of endolymph in both of these tests should produce a nystagmus which would continue for some moments after the endolymph current ceased.

Other phenomena in the rotation experiment are that the maximum after-nystagmus is obtained after about ten rotations, and that a shorter after-nystagmus is obtained when we rotate a patient a shorter or a longer time; and the fact that after a prolonged rotation in certain individuals there is not only a shorter after-nystagmus, but when this dies out there develops a nystagmus directed toward the same side as the turning nystagmus, a socalled after after-nystagmus. These phenomena are not accounted for in Breuer's theory, nor can they be explained with Bárány's hypothesis. They are explained, however, as phenomena of fatigue from overstimulation, if we accept the theory that the duration of the nystagmus is dependent upon the duration of an endolymph current.

ORIGIN OF LABYRINTH TONUS.

The phenomena of labyrinth tonus can be intelligently understood only by keeping in mind the following facts in the physiology of the semicircular canals: (1) An endolymph current stimulates only the hair cells on the side of the crista receiving the impact; (2) an endolymph current in one direction in a canal produces stimulation of the muscles which direct nystagmus toward one side; an endolymph current in the opposite direction stimulates the muscles which direct nystagmus toward the other side. In each semicircular canal the stimulation which results from an endolymph current which directs nystagmus toward the same side is greater than the stimulation from the endolymph current which directs nystagmus to the other side. In other words, in each crista impulses from the hair cells on one side produce nystagmus in one direction, and impulses from the hair cells on the other side produce a nystagmus in the opposite direction, the stronger impulses coming from those hair cells which direct nystagmus toward the same side. Labyrinth tonus is the result of impulses constantly emanating from the hair cells on both sides of each crista. From each labyrinth, therefore, tonus impulses emanate which tend to produce nystagmus toward both sides; but since the stronger impulses are those which produce nystagmus toward the same side, the tonus from a labyrinth, if unchecked by corresponding impulses from the opposite labyrinth, will always produce nystagmus toward the same side. The stimulation of the hair cells which produces tonus impulses is probably occasioned by the intralabyrinth pulsations associated with each beat of the heart. Any unilateral increase in the pulsations would tend to increase the tonus from a labyrinth which would disturb the normal equilibrium and produce a nystagmus directed toward the same side. This condition is observed clinically in cases of labyrinth congestion occasionally associated with acute otitis media; also in most cases of serous labyrinthitis where the congestion of the labyrinth appears to be the most important change. In the severer types of serous labyrinthitis it has been observed that during the onset and during the later stages of recovery there is a spontaneous nystagmus directed toward the same side, produced apparently by hyperemia in the labyrinth. During the height of the process, when the serous exudate is sufficient to paralyze the action of the hair cells, the labyrinth tonus in this labyrinth is suppressed, and for a time spontaneous nystagmus is directed toward the normal side.

In cases of diffuse suppuration of the labyrinth the spontaneous nystagmus is directed toward the normal side, because now the normal tonus from this labyrinth is unchecked by impulses from the diseased labyrinth.

ORIGIN OF COMPENSATORY TONUS AFTER DESTRUCTION OF THE LABYRINTH.

The disturbance of equilibrium which follows the suppression of labyrinth tonus when one labyrinth is destroyed, rapidly disappears because compensatory tonus develops to take the place of the tonus from the destroyed labyrinth. Tonus impulses to the voluntary muscles have two sources: (1) Tonus from the labyrinth, and (2) tonus from extralabyrinth afferent impulses. The sudden destruction of a labyrinth destroys permanently the labyrinth tonus from this side, and at the same time apparently suppresses temporarily much of the extralabyrinth tonus to the same muscles. In the recovery from the disturbed equilibrium which follows the sudden destruction of a labyrinth, the first process is the return of the extralabyrinth tonus impulses which the sudden shock temporarily suppressed. The next step is a compensatory increase in the extralabyrinth tonus to balance the tonus from the normal labyrinth, and, finally, in this readjustment there often appears to be a compensatory increase in those impulses from the normal labyrinth which tend to produce nystagmus toward the opposite side. It is only in cases of long standing labyrinth destruction, where the tonus impulses from the normal labyrinth which direct nystagmus toward the opposite side apparently increase so as to balance tonus impulses which direct nystagmus toward the same side; in other words, when the impulses from the hair cells on the two sides of the cristæ are equal. When the tonus impulses from the hair cells on both sides of a crista are equal, then the after-nystagmus in the rotation tests will be as strong toward the defective as toward the normal side. This has only been observed in cases of long standing unilateral labyrinth destruction.

DISCUSSION.

Dr. Richard H. Brown believes all of the explanations he has heard, and, so far as he followed Dr. Shambaugh, his explanation, also, presupposes that in rotary nystagmus we have a motion circulation in the canal—take the horizontal canal as an example. If this canal were commensurate with the head, it would be a circle of some four inches in diameter, revolving around its center, but in fact this canal is a little thing of about one-quarter inch or less, and revolving at the end of a radius of some two inches. It seems to him that, revolving in that way, we would have a centrifugal action, the fluid tending to force itself to the outermost circumference and circulating through all of the different canals to the outer part. He would suggest that if some ingenious man could make a pair of glass labyrinths, enlarged, and fill them with fluid containing a floating pigment, then mount them on a bar of perhaps four feet in length, in exactly the same relation, and rotate them, as in a centrifuge, and then stop them, he could observe the actual play of the pigment in the glass index. Thus far the theories advanced have never seemed convincing to him.

Dr. E. R. Lewis, of Dubuque, Iowa, believes all the members present were greatly in Dr. Shambaugh's debt for the presentation of this very admirable discussion. He wished particularly to say one thing. When Dr. Shambaugh presented this matter some year and a half ago, he could not accept as plausible his idea that nystagmus in one direction was caused by the influence of the cells on one side of the crista, and nystagmus in the other direction was caused by the influence of the cells of the other side of the crista. At that time the doctor did not bring out the point that in this conception the cells on one side exceed in their impulse value the cells on the other side; and the objection which he raised at that time, that this theory was untenable when we consider that nystagmus is produced by the influence of the cathode which stimulates all the cells equally at the same time, was, he thinks, valid. But now that Dr. Shambaugh has explained that he considers the cells, for instance, in the anterior vertical canal on the utricular side, of higher value than the cells on the canal side, and the cells on the canal side of the horizontal of higher value than those of the utricular side, the objection which he raised is satisfactorily disposed of. He thinks Dr. Shambaugh's theory not only plausible, but very acceptable.

Regarding what the essayist said about the influence of the extralabyrinthine tonus in comparison with the influence of labyrinthine tonus, he thinks there is a great deal to suggest that the relative value of the extralabyrinthine tonus is very great. Consider, for instance, that while awake, whether in sitting or standing still, the opposing groups of skeletal muscles are evenly balanced in their contractions, by reason of the tonogenetic current from the afferent impulses, but when we commence to lose consciousness we commence to fail to perceive afferent impulses and, coincidentally, our muscle tonus begins to diminish. With the onset of sleep in sitting position, for example, which gives the pull of gravity a chance to exert its influence upon the upright head, the first evidence of that diminution of tonus is the nod. The muscles which have held the head upright unconsciously, by reason of that muscle tonus, are relaxed. It cannot be due to labyrinthine tonogenesis alone, because in the very nature of things labyrinthine tonus impulses must go on while asleep as well as awake. It has to do, therefore, with apperception, and if tonogenesis were overwhelmingly of labyrinthine origin, that is, if the amount of tonus impulses emanating from the labyrinth overwhelmingly outmeasured those emanating from other extra-labyrinthine sources, we would not find muscle tonus beginning to diminish when we commence to lose consciousness, and atony commensurate with and coincidental with unconsciousness.

Dr. George W. Boot said there is one law in nystagmus which he has never seen mentioned, and which will help in remembering the directions, namely: Nystagmus is always in the opposite direction to the current in the endolymph. While he thinks Dr. Shambaugh is undoubtedly right with reference to tonus from the labyrinth, namely, that the pulsations of the heart give rise to the greater amount of tonus, he believes that a great deal results from sound waves. The semicircular canals are in such intimate relation with the auditory portion of the labyrinth that sound waves which stimulate the cochlea are almost certain to stimulate to some extent the end organs of the semicircular canals. A good illustration of this tonus is in the action of our muscles when some unexpected noise occurs; for instance, when writing, if an unexpected noise occurs, there is a break in the writing. It is the same phenomenon which makes you hold your breath when you see a person walking in a particularly dangerous place. You do not make a sound, because if you did the sound waves would increase the tonus of his muscles, and the greater contraction of the stronger muscles would disturb his balance and cause your friend to fall.

Dr. J. Gordon Wilson was sure that the members all agreed in acknowledging their indebtedness to Dr. Shambaugh for the interesting discussion and the way in which he placed before them some of the problems involved in the physiology of the labyrinth. His only regret is that the essayist tried to give too much. The subject bristles with difficulties which one would like to discuss at length. Many questions suggest themselves.

For instance, the one raised by Dr. Boot—the relation of hearing to the tonus labyrinth. There is no doubt at all, in Dr. Wilson's mind, of this relationship. He has frequently observed in animals, after one labyrinth had been destroyed, that stimulation of the other ear by sound waves would immediately cause that animal to show a reaction indicating stimulation of the labyrinth. We are all agreed that the labyrinth is one of the most important peripheral organs for tonus, but at present he finds great difficulty in accepting Dr. Shambaugh's hypothesis regarding its origin. In discussing tonus it ought to be made very clear that we know tonus chiefly through its increase or diminution; and that of the tonus which exists when the body is at rest, the continuous tonic reflexes, our knowledge is very indefinite. We know that mechanical irritation of the labyrinth and of its nerves will produce alterations of tonus. Also, from observations on the muscles—especially the eye muscles—we must conclude that when the body is at rest there is also an influence constantly going from labyrinth to these eye muscles. But where does this tonus originate, and how is it caused? Brauer believes it originates in connection with the otoliths. Ewald believes it is produced by cilia movements. There is also the physical pressure theory, under which head one may class such an hypothesis as that of Sydney Scott, who looks for an explanation in the heart's action. To all these, serious objections can be urged, which it would take too long to discuss. At present we must conclude that as to how and where this permanent reflex is produced we have not sufficient knowledge.

Dr. Shambaugh suggests that the hair cells play an important part in the production of tonus by pull or by pressure. We do know that in the horizontal canal of birds, movements of endolymph towards the ampulla cause movements opposite to and greater than those from ampulla to canal. But this exists only for the horizontal canal, and is the reverse in the superior. We would require to suppose, on Dr. Shambaugh's hypothesis, that reversal has occurred in the structure of the ampullæ in the two canals, of which we have no atatomic knowledge. An easier explanation would be an hypothesis in regard to the nerve distribution to these parts along lines he has indicated elsewhere.

Dr. Wilson cannot agree with Dr. Shambaugh that the compensatory action of the tonus comes from the other ear. Rotation after one side labyrinth destruction speaks against such a hypothesis. In animals—for instance, the dog—destruction of the labyrinth results in definite torsion of the head, which is permanent, though the animal recovers from the destruction nystagmus in two or three days. So there are certain muscular actions for which he never compensates, and some for which he does. Now, if the other labyrinth is destroyed, within a short time the head readjusts itself. Here the second labyrinth has not compensated, but may have played some part in preventing the compensation. In such cases he cannot see how the phenomena are to be accounted for by a compensatory mechanism lying in the semicircular canals. On the other hand, positive evidence pointing to the cerebrum as being the seat of the compensation has been given in a recent paper, by Wilson and Pike (The Effects of Stimulation and Extirpation of the Labyrinth of the Ear and Their Relation to the Motor System), read before the Royal Society of London in June, 1912, and published in their Transactions.

There are one or two points in all discussions on the physiology of the labyrinth that ought to be clearly understood. One is in regard to the socalled currents of the endolymph. It is unfortunate that the word current is used for movements of the endolymph in the semicircular canals. Currents are unthinkable. What probably occurs is an alteration in pressure. This view finds expression in the now generally accepted Brown-Mach-Brauer hypothesis.

Again, what comes primarily from the labyrinth is not the nystagmus or double movement, but a deviation, which corresponds to the slow phase in the ocular movement. The quick phase in the ocular movement is extralabyrinthine, and one of the points still in dispute is where it originates.

The paper by Dr. Shambaugh was full of information, and its interest increased by the fact that he drew most of his references from man, the ultimate test to which all our experimental work must be applied. In animals we can perform more definite lesions than is possible in man, and, watching their outcome, can arrive at data which we can apply to pathologic conditions in our special work. We strive to bring these experimental and pathologic conditions into agreement. Whether they agree or not, we are eyer hopeful that the outcome will be a clearer conception than we now have of the physiology of the labyrinth.

Dr. Boot said that both Dr. Shambaugh and Dr. Wilson spoke of the greater sensitiveness of the cells on one side of the crista, but did not offer any suggestions as to why this should be. Bárány has given a possible explanation, namely, that the nerve supply on one side of the crista may be greater than on the other.

Dr. J. Holinger mentioned some experiments published more than ten years ago by Dr. Fischer, in connection with what Dr. Wilson said. Dr. Fischer destroyed one labyrinth completely in a dog. The dog was very sick for a few days, but recovered after about six or seven weeks. Then the other labyrinth was destroyed. After this the dog was sick again, though more seriously, and recovered much more slowly than from the first operation. Then he took other dogs and destroyed a certain part of the cortex of the brain, behind the Rolandic fissure. The dog showed no direct result from this destruction. When, however, in this dog he destroyed one labyrinth, the dog did not recover, and was unable to stand on his feet again. It would have died of inanition if it had not been fed artificially. That would mean that this part of the cortex is capable of taking the place of one labyrinth, because if that part of the cortex was not destroyed the dog would recover. How does that agree with the theory of peripheral tonus?

Dr. Shambaugh, in closing, said that Dr. Brown raised the question, whether the centrifugal force might not work to check a motion of the endolymph in the canals, especially since these canals are located at some distance from the pivot of rotation. This question has been brought up before, and he believes there is no difficulty at all in accounting for the motion of the endolymph in a mechanism of this sort. Dr. Brown also suggested the construction of a model to demonstrate exactly what the reactions are in the semicircular canals. This, of course, is the ideal, if it were only possible to construct such a model; but even if it were possible to devise a model that would resemble approximately the semicircular canal mechanism, such a model would be lacking in the finer membranous structures which no doubt influence decidedly the physical reactions resulting from rotation. He cannot see how one could possibly construct a model that would be of any assistance in this problem. The same question has come up in connection with the action of the membrana tectoria, and it has been suggested that one must devise a model which will demonstrate how the membrana tectoria responds to sound impulses. Of course, it is out of the question to construct such a model, since, in the first place, we don't know exactly what the physical properties of the membrana tectoria are, and, in the second place, even if we did know what these properties are, the mechanism is entirely too delicate and intricate to permit of imitation. Ewald at one time constructed a model in which he placed a taut rubber membrane, which was devised to illustrate the response in the membrana basilaris to the impulses of sound waves. This glass model permitted Ewald to photograph the vibrations of this rubber membrane, as it responded to tones of different pitch. Ewald argued that this illustrated the manner in which the membrana basilaris responds to sound waves. This work of Ewald's attracted a great deal more attention than it deserved. There is no more resemblance between the model constructed by Ewald and the membrana basilaris than between a stretched sheet and strings of a piano.

Dr. Boot and Dr. Wilson suggest that the sound waves entering the ear may pass up through the vestibule and thus bring about a stimulation of the hair cells of the crista, and account in this way for the origin of labyrinth tonus. It would seem that the close anatomic relation between the two parts of the labyrinth might suggest that the same impulses could stimulate the two sets of end organs. It would be interesting if experiments could be devised that might demonstrate this. He does not see that the observation pointed out by Dr. Boot, that a sharp sound startles a person, is necessarily a proof that this is the result of the increased labyrinth tonus.

Dr. Lewis suggests that extralabyrinth tonus impulses are more important than the tonus from the labyrinth. It would seem a priori that the tonus impulses emanating from a mechanism that has been set aside for the purpose of supplying tonus would be more delicate and more important than the more primitive tonus impulses that emanate from other sources. He does not know of any way by which it is possible to determine whether the labyrinth or the extralabyrinth tonus impulses are the more important.

Dr. Lewis asks whether he believes that the principal function in the semicircular canals is to give tonus? Dr. Shambaugh is inclined to believe that the tonus function of the semicircular canals is as important as any function they may have.

Dr. Lewis thinks that the chief function of this whole apparatus is to sense motion, and that tonus is only a secondary function. He asked Dr. Shambaugh whether he does not believe that the extralabyrinth tonus impulses disappear with loss of consciousness?

Dr. Shambaugh does not see any reason why all the extralabyrinthine tonus impulses must necessarily stop with loss of consciousness, especially if these extralabyrinth tonus impulses have their origin in the action of the viscera, such as the pulsation of the heart and the act of respiration.

Dr. Lewis suggests that the nodding which occurs when one falls asleep in a chair is an evidence that the extralabyrinth tonus disappears with loss of consciousness.

LARYNGOLOGICAL SECTION OF THE ST. LOUIS MEDICAL SOCIETY.

Meeting of January 29, 1913.

Paper: Widening of the Palatal Arch for the Correction of Deflections of the Nasal Septum.*

Dr. Geo. V. I. Brown.—The results of clinical and experimental observation, which were presented for consideration by Dr. Brown, were designed to emphasize the possibility of a very much more extended association between defective nasal and maxillary development, with their consequent pathologic states and influences, and general development, trophic changes, nervous disorders, as well as other more or less remote affections, than is commonly recognized in this relation. Certain features of the etiologic, pathologic and structural considerations which bear upon the influence of these oronasal factors in disease are well understood. Other causal factors, though recognized as important to pathologic manifestations in other regions, are not generally accepted as being of nasal and maxillary origin. A third division must, for the present at least, depend to some extent upon the more or less theoretic application of certain developmental features, connection with which, in the absence of definite knowledge, can be established only by comparative consideration of certain vital phenomena which, viewed in the light of experimental results, clinical manifestations, and the more or less arbitrary application of better understood laws governing bodily growth and metabolism, appear to share at least a measure of interdependence.

To establish unrestricted nasal respiration where the nose is narrow, does not give the necessary breathing space. Dr. Brown practices the rapid separation of the superior maxilla at the median palatine suture, the procedure being accomplished in a period of ten days to two weeks. He stated that clinically we have proven that there is an increase in the size of the nares with corresponding tendency toward improvement in the form of the septum and in the reduction of the hypertrophic conditions which represent the pathologic results that are the invariable evidence of such malformations, and that these take place with almost mathematical regularity when the upper maxillæ have been forced apart by separation through the median palatine suture without objectionable local or general disturbance. Early treatment of this character can do much to prevent such defects. The nasal processes of the superior maxillary bones are forced apart when these bones are separated by direct pressure across the palate, and they carry with them, to some extent at least, the attached nasal bones. Thus there is an actual increase in the internal measurements of the width of the nose. It also makes possible a separation of the ridges which form a groove for reception of the vomer. This allows the natural resiliency of the septum to take advantage of the additional space thus afforded, with corresponding tendency toward straightening of its curvatures.

In the discussion it was brought out by Dr. Summa and Dr. Lischer that as a whole the orthodontists question the procedure of opening the median maxillary suture. They are of the opinion that the widening of the palatal arch does not result from opening of the suture, but that it is due to the stretching of the thin plate of bone between the maxillary suture and the alveolar process, and that widening of the nasal tract may be due to slight opening of the suture plus stretching of the palatal process of the maxilla. To get the best final result the occlusion of the teeth must be harmonized. The orthodontists take the stand that it is a mistake to carry the upper arch bucally first and then expect an orthodontist to bring the lower in line.

ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY.

Vol. XXII. JUNE, 1913. No. 2.

XII.

REMOVAL OF ADENOIDS BY DIRECT INSPECTION.*

By Joseph C. Beck, M. D.,

Chicago.

After developing this method of operation for more than a year, I wish to present a brief and concise description, in order to place it on record, hoping that this procedure will so appeal to the operators who are engaged in this field of work that they will give it a trial.

It has always seemed to the writer that the removal of the adenoids, which is such an important operation to the well being of the individual, and so essential for the laryngologist to master, has been performed in an uncertain and unsatisfactory manner. Not that the operation as performed by the majority of men is not followed by good results, but that in a fair percentage of cases, even when performed by the expert, it is followed by what is regarded as recurrences or remains. Especially is this true of that group of adenoid vegetations in the region of the eustachian tubes. The one great difficulty is the inability to see clearly this important region during the operation. While operating for the removal of an epipharyngeal fibroma on a patient who had a short time before had his tonsils removed, I discovered what a large field of the postnasal space was exposed by the method I am about to describe, and it was at that moment that I made up my mind to apply it as a routine in adenectomy.

---

*Read before the Middle Section of the American Laryngological, Rhinological and Otological Association, St. Louis, February 22, 1913.

Dr. E. Pynchon, of Chicago, some four years ago demonstrated to me the retraction of the soft palate by means of rubber tubing, for the purpose of doing my operation of obliteration of the pharyngeal end of the eustachian tube in connection with the cure of chronic suppuration of the middle ear (patulous tube after radical mastoid). I was not able to bring the ostium into very clear view, but I was enabled to employ a large laryngeal mirror and do the work very easily by the indirect method. (Fig. 6.) The great difference of exposure of this region after the tonsils (tonsillectomy) are removed is apparent from the comparison of the two illustrations. (Fig. 5 and Fig. 3.)

The technic of the operation is as follows: Pass the free ends of a rubber urethral catheter (the smaller sizes), one through either nostril, and withdraw them from the mouth, thus crossing the columella of the septum. Put a gauze sponge at this point to prevent too much pressure on the columella. Allow them to remain in this position while the tonsils are being removed, usually placing them to one side or the other out of the field of operation of the tonsillectomy. As soon as one tonsil is out, the end of the catheter corresponding to the side from which the tonsil was removed is drawn taut out of the mouth and over the cheek, while the other end is held. This will bring the pressure from behind forward against the hard palate, and thus stop the greater portion of the bleeding and enable one to do the opposite tonsillectomy with very little interference from bleeding of the tonsil removed. It will also bring the anterior and posterior pillars in apposition, thus closing in the large cavity created by the tonsillectomy. If the bleeding is profuse, place a small gauze sponge between the two pillars and then by a fine rat-tooth forceps (Tuffier) hold them together, also taking in a small bit of the gauze so that it cannot fall out at the bottom. (Fig. 3.)

When both tonsils are excised and the bleeding perfectly controlled, the soft palate is allowed to recede, the two pillars liberated by the removal of the Tuffier forceps and the gauze sponge removed. The assistant then pulls both free ends of the catheter out over the cheeks, causing large exposure. (Fig. 3.) The head is now extended, and the region of the ostia, as well as the highest point of the vault of the pharynx, is seen. One must not look from the side to get the best view of the tubal ends, but straight towards the vault. With a large laryngeal mirror inspection is made of the posterior nares with the entire nasal contents, and the overriding of the adenoid mass into the posterior choanæ is noted. (Fig. 6.)

The mass is now palpated so as to determine the consistency and the amount of overriding. If the patient is not completely anesthetized, or his pharyngeal reflex not yet completely abolished, the examining finger gains the same impression as when examining the postnasal space of a conscious patient, namely: the constrictor muscle fold (Wulst) many times in the form of an incomplete ring. As long as that fold forms, the removal of the adenoids should be deferred and patient completely anesthetized, because this muscle fold may be cut and produce symptoms of stiffness of the neck to which Fein recently called attention, but did not explain on this basis.

The removal of the adenoid mass itself may be done by any method preferred by the operator; but the removal of the remaining portions, especially in Rosenmüller's fossa, should be performed with a straight ring curette, such as it is customary to employ in the curettage of the endometrium. It may be bent to suit any particular shape desired. The pronounced oozing that results from the removal of the central mass is controlled by a gauze sponge mounted on a six-inch artery forceps, by exerting firm pressure for a few moments. This is particularly easy of execution, as the soft palate is drawn out of the way. If after removal of this sponge pressure the oozing does not cease, it may be checked by putting on from one to six artery forceps, grasping a small amount of the underlying soft fibrous tissue that is always present after the removal of the adenoids. (Fig. 4.) These are allowed to remain for a few minutes, or until the time the patient awakes from the anesthesia.

The instrument that I employ now for the removal of the main mass is an adenoid curette of the St. Clair-Thompson pattern, without a guard, and with the cutting edge formed into a wave blade. (Fig. 9.) The motion of the instrument in the removal of the mass is not in the usual rapid, sweeping movement of the elbow-wrist turn, but is from side to side and slow elbow-wrist turn. The mass is cut off and remains lying practically in situ; however, the last cut at the lower margin of the mass may require the use of a forceps to hold it. Shredding of the mucous membrane is not possible under these circumstances, nor it is possible to cut off any of the muscle fibers of the constrictor fold, as I have seen in the operation when done by feeling alone.

There are two other points in the operation that are, in my opinion, important, namely: the method of administration of the anesthetic, and the employment of suction to remove secretion and blood. I employ ether exclusively after having tried gas-oxygen for some time, as I find that the latter does not completely relax the pharyngeal muscles, which is necessary for the performance of tonsillectomy and more so for adenectomy, as will be shown later. To induce the anesthesia we employ what Prof. Rovsing of Sweden recommended: the rebreathing of ether by means of a somnoform apparatus. (Fig. 10.) About two ounces of ether is placed in the rubber bag, and after applying the mask firmly over the nose and mouth, the air valve is opened and the patient breathes air and a small amount of ether. When the air valve is partly closed, the ether vapor is increased until finally the valve is entirely closed and the patient breathes pure ether. This he continues to do until finally a quantity of carbonic acid will form within the bag. This is called the rebreathing of ether, since the patient exhales some of the excess. The carbonic acid is considered of benefit to a certain degree in ether anesthesia. Whenever it accumulates to an excess, cyanosis is present; the air valve is opened and some air is allowed to enter. This method of induced anesthesia we have found to be the least irritating and most satisfactory, although it requires a little longer to induce anesthesia than by other methods. The induced anesthesia is then followed by the forced vapor method.

The apparatus illustrated (Fig. 11) shows an ether bottle attached to a pump, run by a small motor, whose speed can be regulated (the Victor transformer cautery apparatus). This vaporized ether is forced through a small tank (Cain McDermot) which contains wound within it tubing surrounded by thermolite. This tank is hermetically sealed, and when placed in hot water for about ten minutes heats up the thermolite; the latter holds the heat for some time. The ether which is passing through the heated tubes within this tank thus has its chill taken off and is easier of respiration, absorption and less irritating to the mucous membrane of the respiratory tract. From this tank pass the ether supply tube and its terminal mouthpiece. This latter acts also as a cheek retractor for better inspection. There is a suction bottle for the blood and mucus. From the pump, which has a double action of suction and pumping, a stiff rubber tube passes to the suction bottle, and connected with the latter is another piece of rubber tubing with a metal tube to be introduced into the throat, etc., whenever indicated. Suction is one of the greatest aids in the operation of tonsillectomy and adenectomy. Anyone having a motor may use the pump by obtaining the proper attachment.

In conclusion, I wish to say that in tonsillectomy, whatever method may be adopted, it is necessary that the operation be performed as rapidly as possible in order to utilize the pressure by the catheters to control bleeding. The operation as first suggested by Dr. Sluder, of St. Louis, or that one by Dr. J. Leslie Davis, of Philadelphia, is such as meets the occasion. The method that I recommend* and that I am able to use almost in every instance, is the one that gives me the best satisfaction in connection with this method of operating on the adenoids. I take the liberty of presenting two illustrations (Figs. 1 and 2) of my method of doing tonsillectomy without preliminary dissection, because the removal of the tonsils and adenoids, especially in children, is usually combined or performed at the same sitting.

I have not changed my technic of tonsillectomy from the one originally described, except in so far as it relates to this adenoid operation. I take exception to the criticism of Dr. Greenfield Sluder in the *Journal of the American Medical Association,* March 1, 1913. He states:

"In Beck's instrument the narrowing gap which is to do the cutting is a snare-wire loop, concealed and protected in the Vedder tip. As soon as the snare is pulled the loop is released from its rigid protector. It must then be trusted to follow in the connective tissue bed. Frequently it does follow nicely, especially if there are no adhesions between the capsule and the subjacent tissue. But should it not do so, the surgeon has not the power to tuck the tonsil into it, because he has nothing rigid to resist his pressure; the flexible wire bends with the tissues. He, therefore, has no rigid ring over which to pass the finger tip in forming his final judgment of when to cut. For these reasons the repair work which may frequently be necessary cannot be done, especially the removal of thin tough pieces, which is quite possible, usually, with the original model. A small finger tip is also a disadvantage for Beck's instrument. In other respects it is admirable, possessing the advantage of the slowly strangulating snare."

This can only occur when the operator is not entirely familiar with the technic. If once the tonsil is Sluderized (pushed through the ring of the tonsillectome) and the finger is pressed until the entire circumference of the ring is felt under the palatoglossus muscle, the wire is under constant control and will follow the contour of the tonsil and remove it in its entirety, with a smooth uninjured capsule and nothing else. Even the plicæ tonsillares, superior and inferior, remain, which is not always possible with the blade going from before backwards.

---

*Tonsillectomy by Means of a Single Instrument, the Tonsillectome. Journal of the American Medical Association, June, 1912.

The one modification that I have made is to have a much smaller ring in cases of very flat tonsils and small tonsils of children, since a perfect operation depends on the entire cryptic surface of the tonsil being engaged in the ring.

I wish to illustrate a case of attempted removal of the adenoids, which evidently were only severed at one place and allowed to hang on and organize in the form of a polyp. (Figs. 7 and 8.) Such an accident could not occur if the operation had been performed, even by a novice, by direct inspection, as I have described.

Since writing the above, the author has been in personal communication with Dr. George L. Richards (Fall River, Massachusetts), who referred him to his paper as published in the Transactions of the American Laryngological Association, 1907, page 184, in which he stated his practice of using the palate retractor. In justice to Dr. Richards, his description is appended.

"In young adults and older children, I have used a palate retractor first suggested to me by Dr. J. E. Schadle of St. Paul, Minn. This consists of a piece of tape twenty-four inches long, the ends of which for six inches are twisted and then coated with paste and dried. One end is passed through each nostril to the nasopharynx, grasped with a forceps and drawn out through the mouth, where it is held by nurse or patient. The tractor draws the palate well forward and allows the instrument to be easily inserted. Here cocain through the nose and by way of the mouth answers for anesthesia."

While acknowledging priority to the Schadle-Richards method of retracting the palate, the author wishes to draw attention to the fact that the object of his retraction is different from that as pointed out in Dr. Richards' paper.

(1) Dr. Richards uses the tape method of retraction in connection with local anesthesia; the author uses it in connection with general anesthesia.

(2) Dr. Richards' object is to provide ample space for the insertion of the adenectome; the author's object is to operate in the nasopharynx under direct inspection, especially in the region of the eustachian orifices.

(3) Furthermore the author makes use of this method as an aid in the management and control of primary tonsillar hemorrhage by means of drawing the posterior pillars over the tonsillar fossæ.

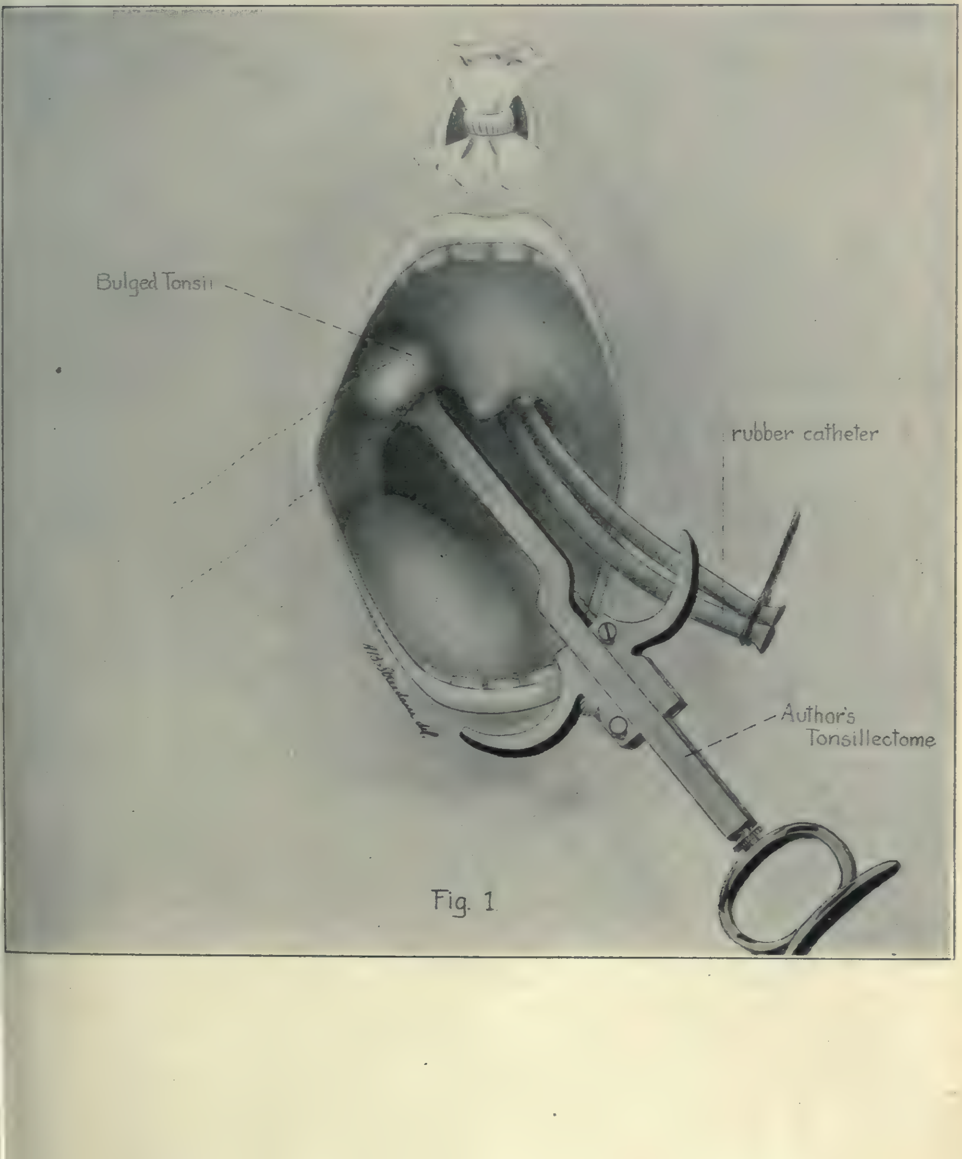


Fig. 1

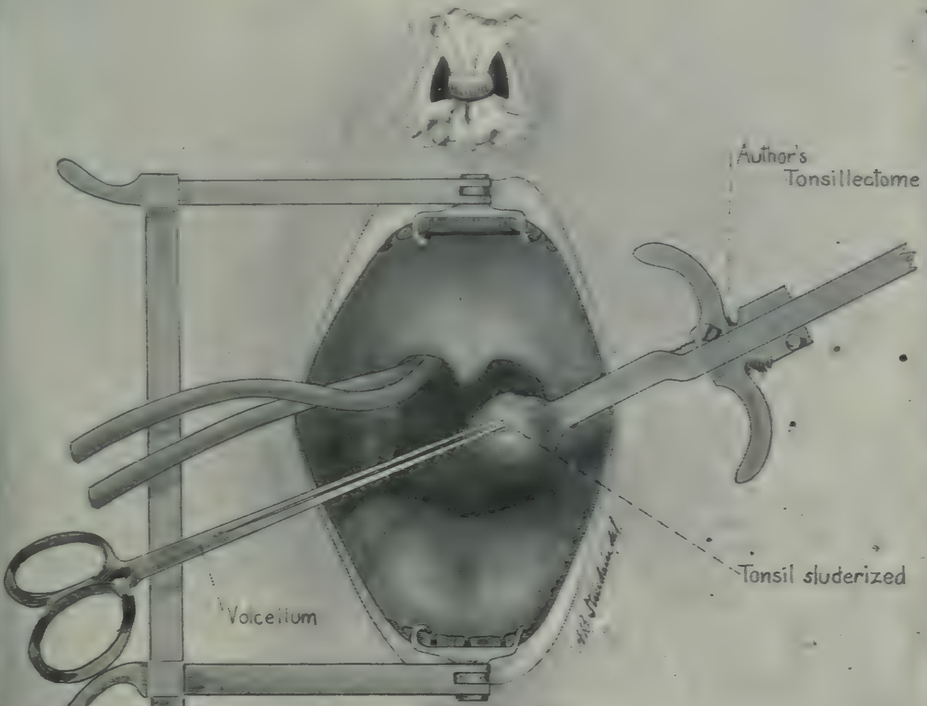


Fig. 2

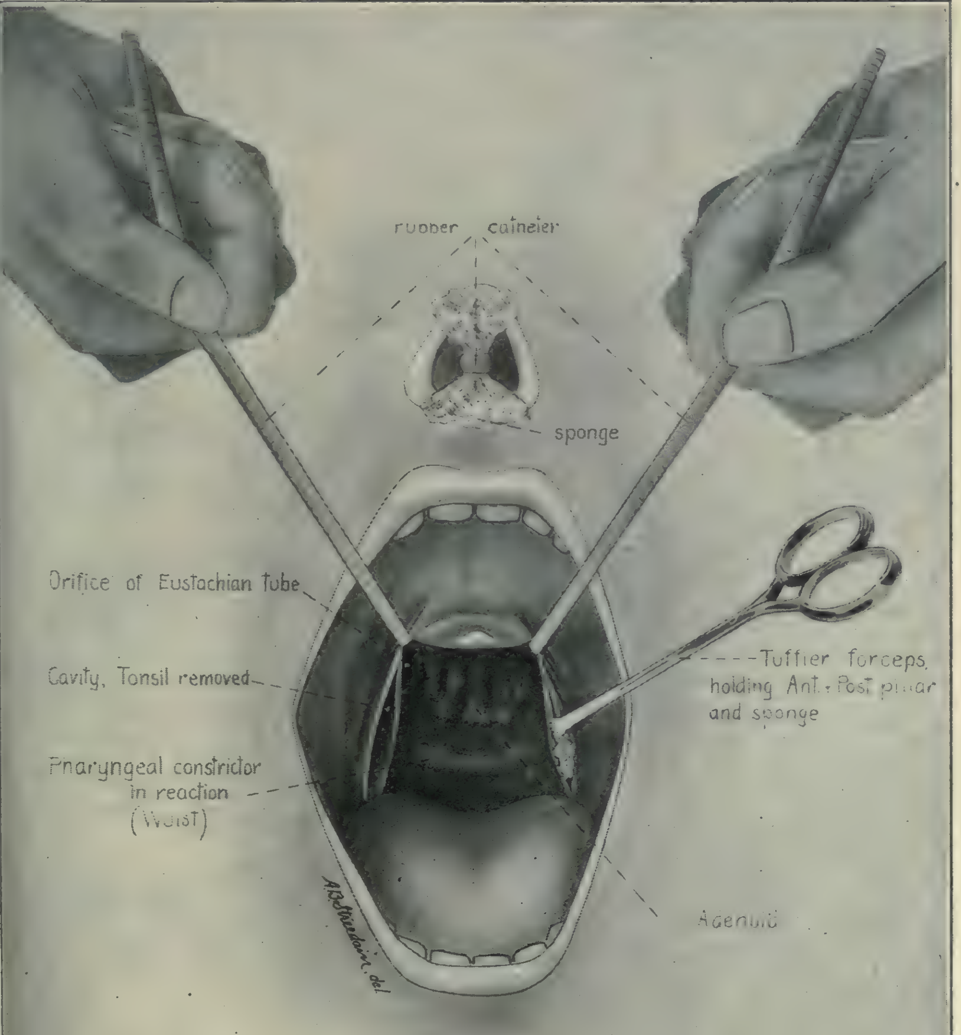

rubber catheter
sponge
Orifice of Eustachian tube
Cavity, Tonsil removed
Pharyngeal constrictor in reaction (Waist)
Tuffier forceps, holding Ant. + Post. pillar and sponge
Adenoid
A.B. Striedain, del

Fig. 3

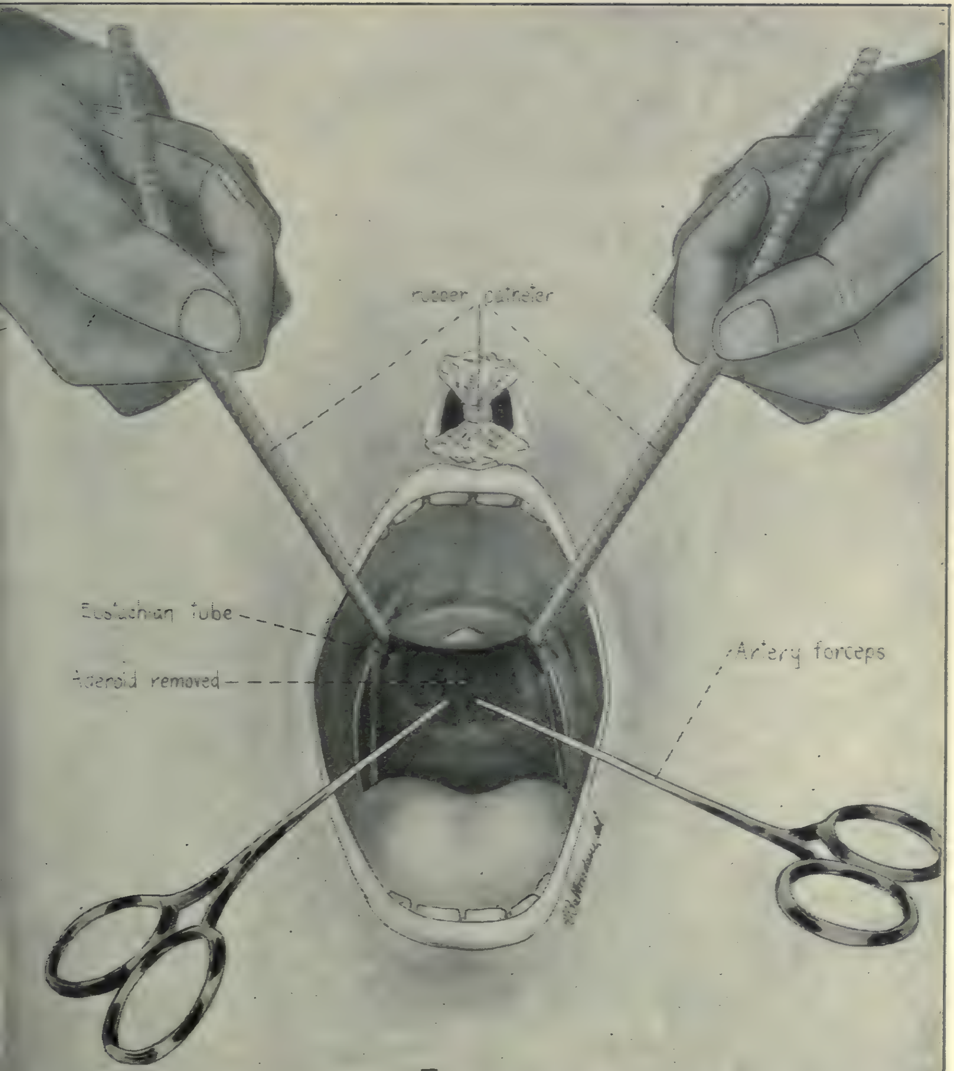

rubber catheter
Eustachian tube
Adenoid removed
Artery forceps

Fig. 4

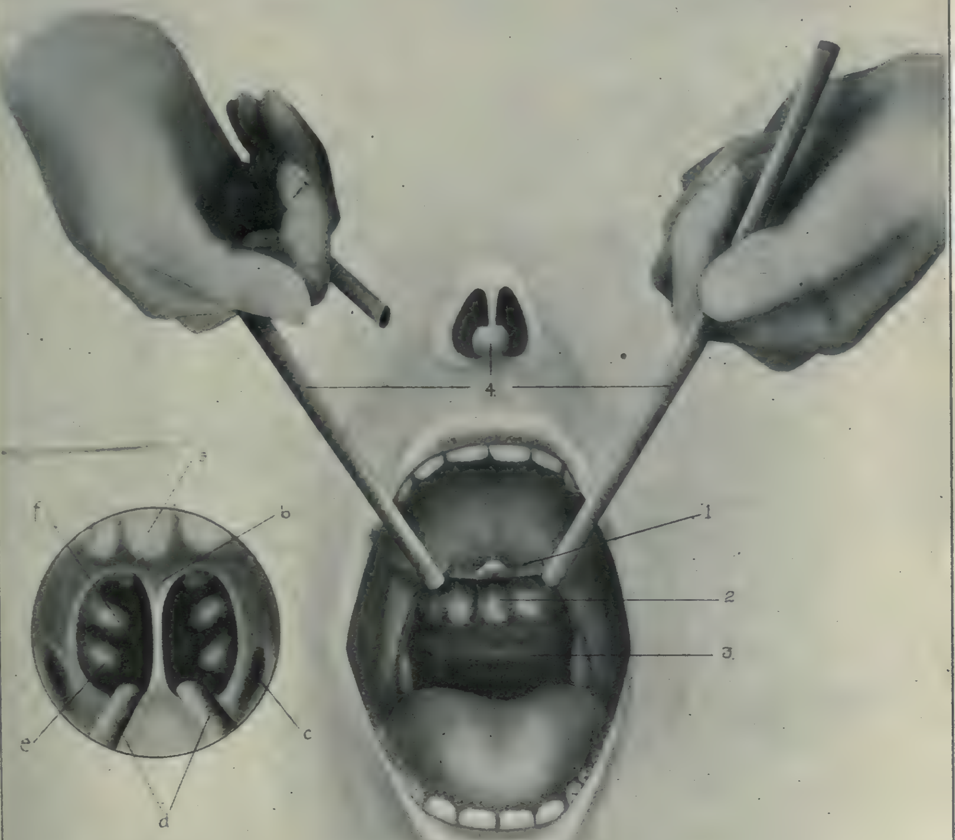


Fig. 6

A. B. Streedain del.

Fig. 5.

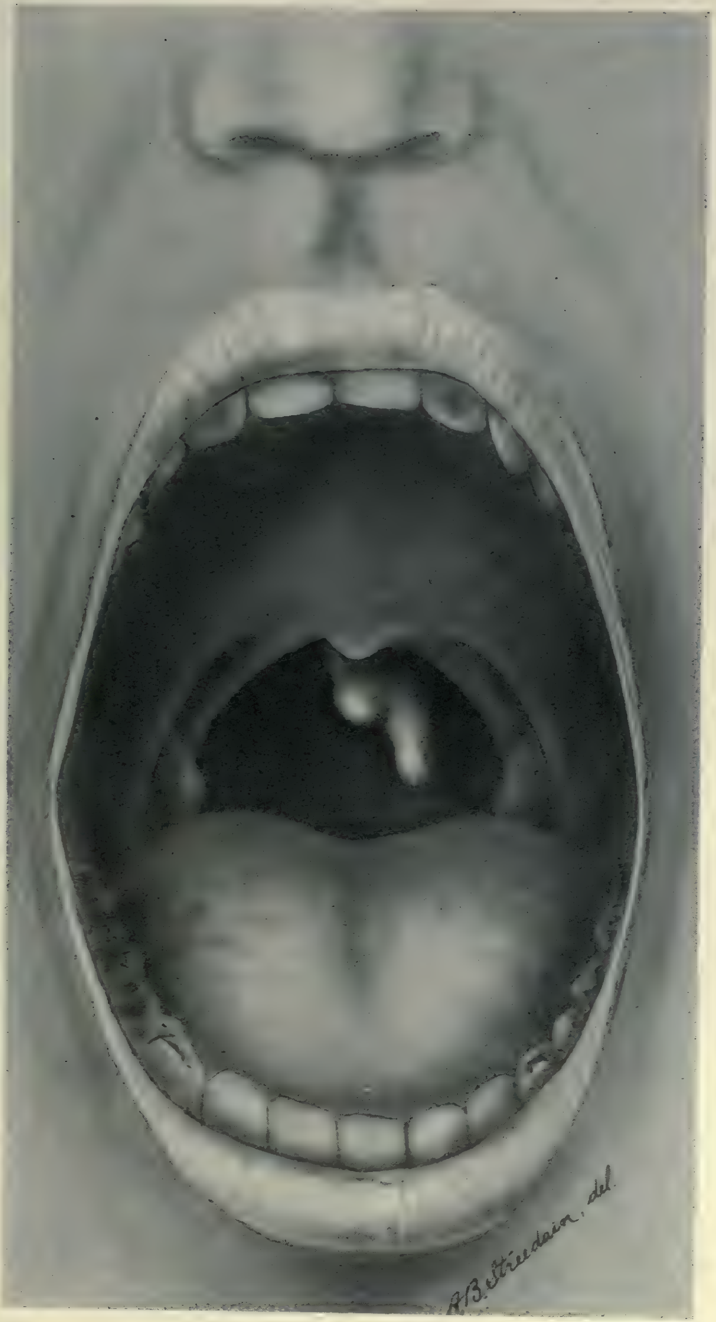


Fig. 7.

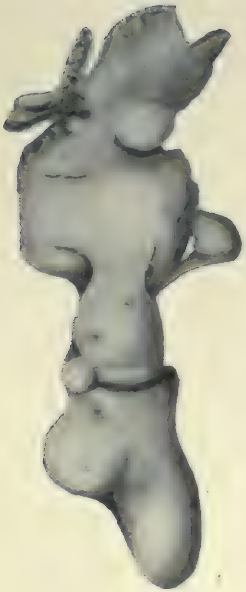


Fig. 8.

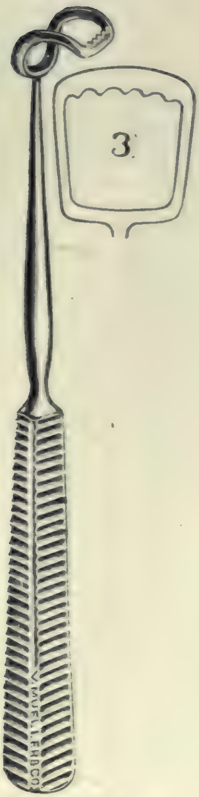


Fig. 9.

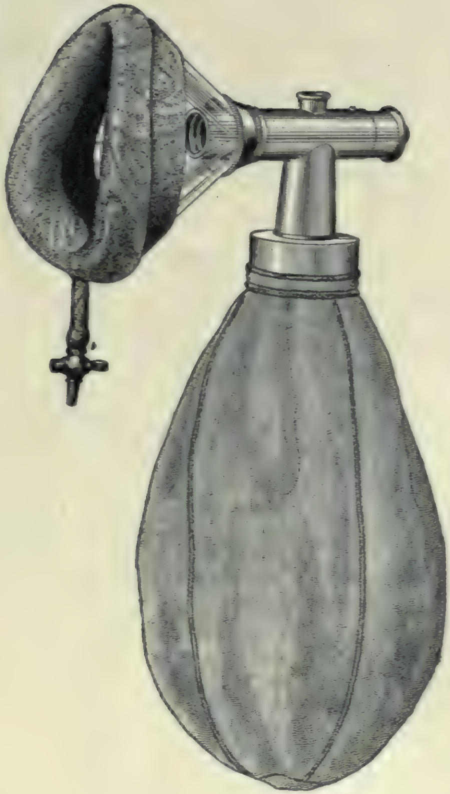


Fig. 10.

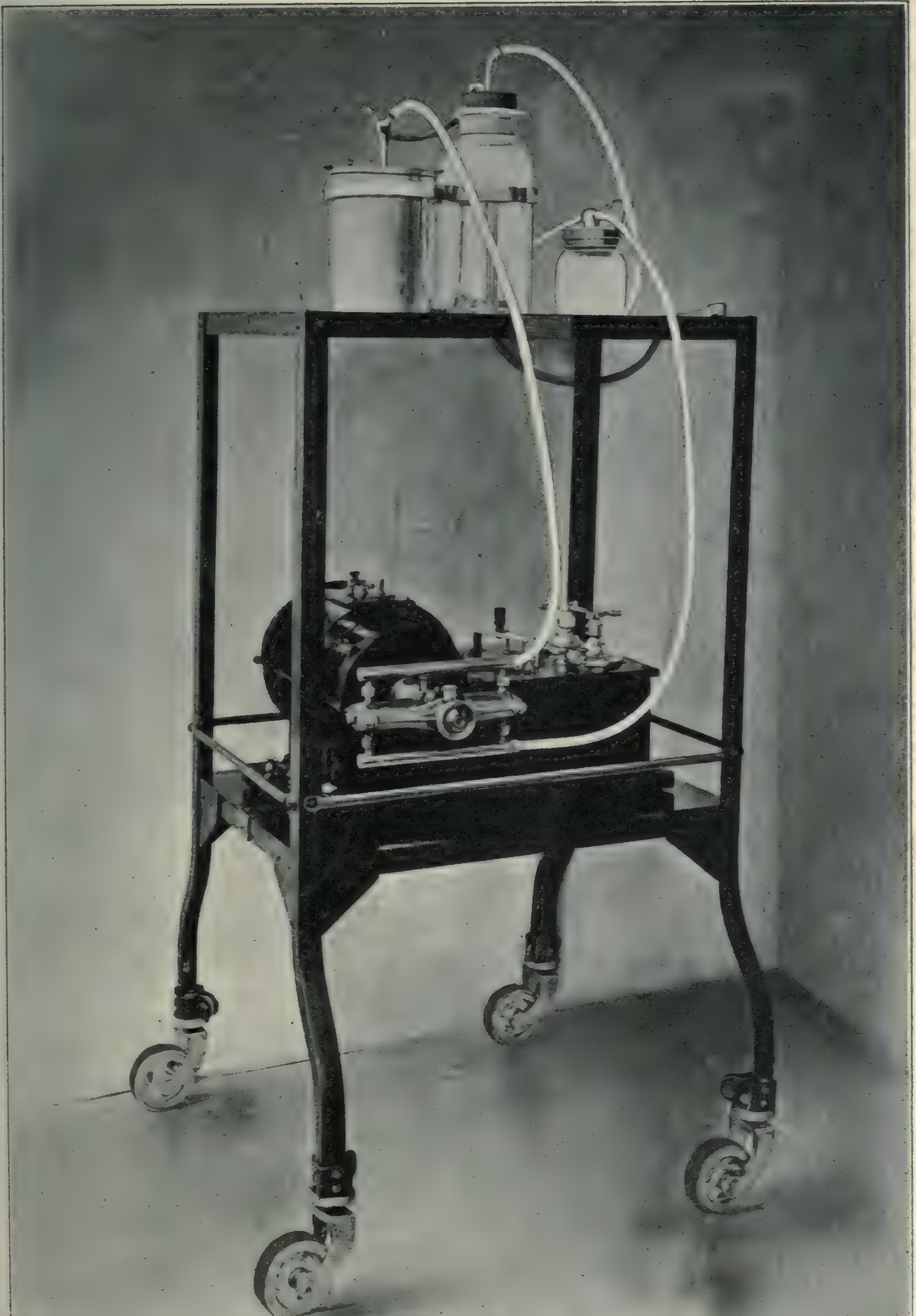


Fig. II

XIII.

WHEN TO OPERATE ON THE LABYRINTH IN LABYRINTH INFECTION SECONDARY TO PURULENT OTITIS MEDIA.*

By G. E. Shambaugh, M. D.,

Chicago.

The most difficult question in the treatment of labyrinthitis secondary to purulent otitis media hinges on the indications for an operation on the labyrinth. A labyrinth operation in cases of labyrinth infection is called for chiefly because of the danger of a secondary intracranial complication. Invasion of the labyrinth in suppurative otitis media presents a varying clinical picture. The process may be restricted to but a part of the internal ear or it may spread throughout the entire labyrinth. The degree of inflammatory reaction in the labyrinth varies from a slight congestion, with slight alteration in the labyrinth fluids and a partial suppression of labyrinth function, to a diffuse purulent condition producing a total destruction of function. The milder cases of acute labyrinthitis, those which stop short of a complete suppression of labyrinth function, are usually cases of serous labyrinthitis, while the severe cases, those which produce a complete suppression of function, are, as a rule, cases of empyema of the labyrinth. In the milder cases, those which produce but a partial suppression of labyrinth function, we expect, as a rule, a more or less complete restoration of function after the subsidence of the labyrinthitis. In the cases of diffuse empyema of the labyrinth there appears to be always a permanent total loss of function, because the delicate sense epithelium has been entirely destroyed. Cases of acute labyrinthitis do occur, however, where there appears to be a complete suppression of function, both of the cochlea and the semicircular canals, and where on the subsidence of the acute process there is at least a partial restoration of function.

---

*Read before the Ninth International Otological Congress, at Boston, August 12-17, 1912.

An operation on the labyrinth is not indicated in the milder cases of labyrinthitis, although Ruttin has shown that such cases may occasionally be followed by a fatal intracranial complication. It is only in cases of diffuse empyema, where the function of the labyrinth has already been destroyed, that the dangers of an intracranial complication constitute an indication for a labyrinth operation.

The conditions under which an empyema of the labyrinth presents itself varies. If the process is observed soon after an acute onset, it will be "manifest" by distinct active symptoms—disturbance of equilibrium and nystagmus. If it is seen first after it has existed for some time, all active symptoms may have disappeared and the process may be distinctly "latent." The labyrinth suppuration may be a sequel to an acute or a chronic purulent otitis media. If the labyrinth suppuration develops in connection with an acute otitis media, it may occur as a part of a violent panotitis, such as occasionally follows scarlet fever or measles; or it may occur secondary to the more subacute stage of otitis media, as the sequel of a neglected mastoiditis. On the other hand, an empyema of the labyrinth may be secondary to an acute otitis media, when neither the general symptoms nor the local conditions about the ear suggest a process which would require a mastoid operation.

When an empyema of the labyrinth develops in chronic suppuration of the middle ear, it may occur in cases where the disease process has invaded the temporal bone, with or without cholesteatoma formation, that is, in cases where a radical mastoid operation is already indicated; or it may develop in cases where there exists no clinical evidence of a process which calls for a mastoid operation. A diffuse suppuration of the labyrinth, moreover, may exist as a simple empyema, or it may be complicated by erosion of the capsule of the labyrinth, by fistula formation into the labyrinth, by facial paralysis, or by sequestration of a part or the whole of the bony capsule of the labyrinth.

The dangers of an intracranial complication from an empyema of the labyrinth developing under these varying conditions is not always the same. For example, the danger from a case of labyrinthitis which occurs as a part of a violent acute panotitis or from a labyrinthitis secondary to a neglected acute mastoiditis, is very much greater than when the invasion of the labyrinth occurs in a milder otitis media without distinct evidence of a mastoid involvement. In the same way, an empyema of the labyrinth occurring in cases of chronic otitis media where there already exists an invasion of the temporal bone or a cholesteatoma, seldom heals spontaneously and is, therefore, much more likely to lead sooner or later to an intracranial complication than when it develops in a case of chronic otitis media where the clinical indications for a radical mastoid operation are absent. An empyema of the labyrinth, complicated by erosion of the labyrinth capsule and fistula formation, facial paralysis or sequestration, seldom heals spontaneously, and is much more dangerous than a simple uncomplicated empyema.

In view of these clinical facts, it is apparent that the indication for a labyrinth operation is much more urgent in certain cases of empyema of the labyrinth than in others. The situation is quite similar to the question of the indication for the radical mastoid operation in the treatment of chronic purulent otitis media. In both conditions grave danger exists of serious intracranial complications. There was a time when this danger in chronic purulent otitis media seemed, at least to some, to constitute an indication in most of these cases for the radical mastoid operation. Otology has now advanced from this position to the place where we are able to make a more or less sharp distinction between those cases of chronic purulent otitis media where the danger from an intracranial complication is such as to constitute an indication for the radical mastoid operation and the cases where this operation need not be done. In the same way there are those today who would advocate the labyrinth operation for all cases of labyrinth infection severe enough to cause a total suppression of labyrinth function. The question arises whether we are able to make a distinction between those cases of labyrinth empyema where the danger of an intracranial complication is sufficient to constitute an indication for the labyrinth operation and other cases of labyrinth empyema where this operation need not be done. Such a question can be settled only by the accumulated experience from the study of a large number of cases of labyrinth empyema. It is well to remember that accurate observation of cases of labyrinth infection is still comparatively new, and that we have as yet hardly reached the stage where definite indications can be laid down to cover all cases. In view of the large number of cases of labyrinth empyema which recover spontaneously, it is extremely desirable to make a distinction between those cases where a spontaneous recovery is extremely unlikely, that is, those in which the danger of a complication is grave, and the cases where the labyrinth operation may at least be delayed with relative safety, awaiting the first evidence of a further extension.

As pointed out above, the labyrinth operation is not called for in cases of labyrinthitis secondary to purulent otitis media, when the function of the internal ear has not been completely destroyed. In this connection it should be remembered that a circumscribed suppuration of the labyrinth may occur which destroys the vestibular apparatus without involving the cochlea, and which may lead to a fatal intracranial complication without at any time destroying the hearing. Such cases should be treated conservatively, unless evidence of an intracranial extension appears, when the labyrinth operation must be resorted to, even though the operation will destroy the remnant of hearing in that ear. The same method of procedure should be applied to all the cases of labyrinthitis which completely destroy the function of the internal ear, whether they occur secondary to an acute otitis media or in connection with an acute exacerbation of a chronic otitis media, provided no clearly recognized indications exist for a mastoid operation. Furthermore, a labyrinthitis which develops after a mastoid operation, even when it results in complete suppression of function, should be treated conseratively unless symptoms develop indicating an intracranial extension.

On the other hand, the cases of labyrinthitis where a labyrinth operation seems to be clearly called for, include:

First, cases of labyrinth suppuration where clinical symptoms exist suggesting a beginning intracranial complication, such as altered cerebrospinal fluid, severe unilateral headache, etc.

Second, cases where the labyrinth empyema develops as a part of a violent acute panotitis, where the indications for a mastoid operation exist.

Third, cases where the labyrinth suppuration develops as a sequel to chronic purulent otitis media, where well recognized indications for a radical mastoid operation exist.

Fourth, cases where the labyrinth suppuration is complicated by erosion of the labyrinth capsule, by fistula formation into the labyrinth, by facial paralysis or by sequestration of a part or the whole of the labyrinth capsule.

XIV.

SOME ATTEMPTS AT THE INTRANASAL TRANSPLANTATION OF NASAL TISSUES.*

(PRELIMINARY REPORT.)

BY SAMUEL IGLAUER, B. S., M. D.,

CINCINNATI.

The free transplantation of tissues has been extensively employed in general surgery, as exemplified especially in the grafting of skin and in the transfer of bone and cartilage.

In the rhinologic literature, on the other hand, there are very few references to this procedure. Marx[1] reports a case in which he successfully transplanted some fat tissue into a defect remaining after a frontal sinus operation. Portions of the septal cartilage, after having been removed in a submucous operation, have been successfully replaced by Goldsmith[2] and by Kerr.[3] Von Eicken[4] has obtained excellent results through Thiersch skin-grafts introduced into the nasal cavities for the correction of synechiæ. The implantation by Flatau[5] of ivory pegs into the nasal cavities, in order to stimulate bone formation in atrophic rhinitis, can scarcely be classified as a true transplantation operation.

Some time ago it occurred to me that there were certain intranasal conditions which might be benefited or relieved by the free transplantation of the nasal mucous membrane, with or without the underlying bone. Thus, in atrophic rhinitis it seemed promising if one could narrow the wide nasal passages by the introduction of normal bone and mucous membrane. The introduction of mucous membrane grafts to prevent the formation of synechia naturally suggested itself. The free transplantation of mucous membrane for the closure of small septal perforations also seemed worthy of trial. Furthermore, all of us have seen the results of some turbinectomy operations in which we devoutly wished that some of the tissue which had been removed might be restored.

---

*Read before the Middle Section of the American Laryngological, Rhinological and Otological Association, St. Louis, February 22, 1913.

Accordingly, in a limited number of animal and clinical experiments, I have undertaken the free transplantation of nasal tissues. Before describing these operations, it may be well to consider some of the general principles underlying transplantation.

Tissues may be transplanted from one part of the body to another part in the same individual, constituting autoplasty; or, the transfer may take place from one animal to another of the same species, constituting homoplasty. Experimental and clinical experience have shown that in all instances autoplasty will give much better, and more certain results than homoplasty. "The more highly differentiated the cells, the less the prospects of successful transplantation. Homoplasty succeeds best with the connective tissue group, such as fascia, tendons, periosteum, bone and cartilage," (Axhausen).[6] Lexer[7] states that epidermis as well as mucous membrane cannot be permanently grafted from one individual to another, while Schoene[8] affirms that skin transplantation may at times succeed between animals closely related, and but rarely between those not related.

Despite the recorded difficulties and failures in the homoplastic transplantation of mucous membranes, I determined to try the procedure. It seemed possible to me that the nasal mucosa might behave differently from other mucous membranes, since its deeper layers are really periosteum which lends itself readily to transplantation. Furthermore, the excellent results recorded with bone transplantation in general made it likely that the submucous implantation of nasal bones would prove efficacious. Two methods of transplantation were employed. The first consisted in surface transplantation upon denuded areas, comparable to Thiersch grafts.

The second procedure was by submucous implantation; i. e., placing the transplant into a pocket formed by elevating the mucosa. In two rabbits autoplastic surface transplantation of mucous membrane containing a sliver of bone was undertaken. In one of the animals the transplanted tissue was found in situ, loosely attached and imbedded, and had apparently survived after two weeks. In the second rabbit, in which the transplant had been made upon the lateral wall of the nose, the graft was found necrotic at the end of twenty-seven days.

In a dog the following experiment was performed: An opening was made into the nasal fossa on one side, and some of the mucous membrane and underlying bone were taken from the turbinate body on the same side. This tissue, about the size of a five cent piece, was immediately transferred to a tunnel formed by elevating the septal mucosa in the same animal. At the end of seventeen days the animal was killed, and macroscopically the transplant was found alive and in good condition.

The microscopic report by Dr. Brown is as follows:

Slide I: The section shows the transplant firmly in place inside the raised mucous membrane. It is very vascular, seems to be in a healthful condition. Signs of survival are present, inasmuch as some of the cell neucleii stain rather well. Spicules of bone and cartilage are present. Mucous glands are in evidence in the transplant.

Slide II: (a) Shows about the same condition. (b) Practically similar with slight indication of growing cells. (Figs. I and II.)

In none of the above is there any sign of infection and no evidence of an atrophy of the transplanted tissue, nor of its conversion into scar tissue.

A similar experiment in a rabbit showed the tissue intact after three weeks. The microscopic findings were as follows:

Slide I: (a) The transplant seems to be firmly in place under septal mucous membrane. The transplant is highly vascular and in most places shows a comparatively dense round celled infiltration in the fibrous stroma. The infiltration extends sharply into areas of partially resorbed bone and cartilage. The tissue on the whole appears healthy, and no signs of infection are present. In one or two places there seems to be a union between the transplanted tissue and the mucous membrane under which it had been placed.

CLINICAL CASES.

I have the following clinical cases to report:

Case I.—Submucous transplantation of a turbinate for the relief of atrophic rhinitis. H. S., aged 20, male, consulted me, November 10, 1910. Chief complaint, dry throat and stuffiness in the nose. Patient had always been well, except for occasional headaches, which he attributed to the nasal condition.

Onset.—About a year before he suffered with very dry throat, worse at night, sense of smell less acute than formerly. Very few crusts in the nose without ozena. Had been treated at two dispensaries for eleven months and had used washes and an atomizer.

Present condition.—Pharyngitis sicca, especially on the posterior wall. The inferior and middle turbinates on the right side are hypertrophied and covered with thick mucus. The right middle turbinate touches the septum. On the left side the inferior turbinate is atrophied so that the pharynx is visible through the inferior meatus. The middle turbinal is hypertrophied, boggy at its lower margin, which overhangs the inferior turbinal and touches the septum. There is a small spur on the septum, which deviates slightly to the right.

Diagnosis.—Simple atrophic rhinitis affecting left inferior turbinate. Pharyngitis sicca.

First operation, November 10, 1910.—An electrocautery was applied to the inferior turbinal on the left side, and to a point immediately opposite on the lower external border of the middle turbinate, with the idea of producing adhesions between the two prior to the transposition of a portion of the middle turbinal into the lower meatus.

Examinations two and four days later failed to show any adhesions. Eight days later the middle turbinal was freed by an incision occupying about one-half of its attachment, causing it to drop down in contact with the lower turbinal. Cautery applied to points of contact and packing introduced to hold the turbinals together. Examination one month later failed to show any adhesions, despite repeated cauterization.

Second Operation, February 25, 1911.—A submucous elevation was made through a small incision on the left side of the septum opposite the inferior turbinate. (Width of the uncocainized nose 8 to 9 mm.) A turbinate which had been removed at the same time from an otherwise normal adult was then shaved down to form a wedge containing a spicule of bone. This wedge was about an inch and a quarter long and a quarter of an inch at its thickest part. It was introduced into the submucous tunnel, with the bone in contact with the septum, and was thus completely covered. A small piece of mucous membrane was also transplanted onto the raw surface of the middle turbinal on the same side. Examination about two weeks later indicated that the transplant had survived in its new situation. One month later there seemed to be some atrophy of the transplant, as the lumen of the inferior meatus was increasing in width. April 20, 1911, the septal mucosa overlying the transplant was incised along the crest of the transplanted tissue, which was thus exposed and found in good condition and firmly united. Subsequent examinations at various times indicated that the transplant was gradually being absorbed. January 22, 1913, examination showed transposed middle turbinate in situ. There is a slight swell at the site of the implantation, but the transplant seems almost entirely absorbed.

Case II.—Surface transplantation of mucous membrane. N. S., male, aged 16. Chief complaint, formation of crusts in the nose. The condition had started several years before with the discharge of mucus from both nostrils, and later with crust formation. Crusts occasionally slide into the pharynx and are hard to dislodge. Gradual diminution in the sense of small; can sometimes notice odor to crusts; occasional hoarseness.

Examination.—Septum somewhat deviated to the right. Right side almost normal. Left side, atrophy of the inferior turbinal so that the pharynx is plainly visible through the nose.

Operation, September 4, 1911.—A portion of the hypertrophied middle turbinate was removed from an otherwise healthy girl of thirteen. After rinsing the specimen in saline solution, it was transferred to a cross-shaped denudation of the septum of the recipient. The denuded area was opposite the inferior turbinal, and a small strap of mucous membrane was left to hold the transplant in position. A light pack was introduced. September 9, 1911, transplant adherent and healthful. Meatus narrowed so that the transplant is in contact with the atrophied inferior turbinal. September 17, 1911, transplant adherent, growing, somewhat smaller than before. September 19, 1911, transplant growing nicely.

Case III.—Free transplantation of mucous membrane onto the surface of a syphilitic stenosis of the nostril. J. S., aged 33, male. Patient had lues fifteen years ago. Nasal obstruction dates back about three or four years. Received salvarsan two months ago. Diagnosis, slight saddle nose. Cutaneous web just within margin of the vestibule, closing the nostril except for a very small central opening. Complete atrophy of the inferior turbinate on the right side, a few crusts on the middle turbinate.

Treatment.—Incision of the cicatrix, followed by the wearing of a metal splint. Some time later an attempt was made to graft mucous membrane onto the denuded area, but the graft failed to hold.

Case IV.—Transplantation of mucous membrane for the relief of simple atrophic rhinitis. W. S., aged 18, male. Chief complaints, hacking cough, crusts in the left nostril and dry throat. The trouble dates from childhood. In April, 1910, I injected paraffin under the septal mucosa of both sides of the nose. This was followed by less dryness in the throat and absence of crust formation in the nose. This improvement continued until a month ago, when some of the paraffin came out from the left side, with the return of some of the previous symptoms.

Examination.—Partial atrophy of lower turbinate on left side with the ridge, due to the paraffin, still present on the right side. Left nasal passage was patent, especially opposite the inferior turbinal. The pharynx, which was smooth and somewhat glazed, could be seen through the nose on the left side.

Operation, July 1, 1911.—Under cocain anesthesia a cross-shaped incision one and a half inches long, similar to Case II, with a strap of mucous membrane to hold the flap, was made along the septum opposite the left lower turbinal. A piece of hypertrophied mucous membrane was removed from the middle turbinate of another otherwise normal male patient. The transplant was washed in normal salt solution and then placed on the denuded cartilage under the strap. The cavity was lightly packed with gauze, which was removed thirty-six hours later and showed the flap in place (apparently). July 8, 1911, transplant is growing nicely, some granulations along the line of incision. Transplant is in contact with the lower turbinate. Patient states that throat is not as dry as before operation.

About one year later the transplant was still present, but had shrunken somewhat. January 25, 1913, about a year and a half after the operation, the site of the transplant is still evident, but it does not stand out as a ridge.

Case V.—Transplantation of bone and mucous membrane into the septum for the relief of atrophic rhinitis. A. R., aged 18, male. Complains of headache, ringing in the ears, and throbbing in the head. The trouble began three or four years ago; has crusts in the nose and has lost the sense of smell.

Examination.—Atrophy of right lower turbinate. Hypertrophy of the right middle turbinal with small polypi anteriorly. Marked atrophy of the left lower turbinal, leaving the meatus wide as a lead pencil, with pharynx plainly visible through the nose. Creamy pus in both nostrils, especially surrounding the polypi. No ozena.

Diagnosis.—Atrophic rhinitis with ethmoiditis on the right.

Operation November 30, 1912.—Submucous elevation along the septum on the left, forming a tunnel about one and one-half inches long and about one inch wide. (Cocain-adrenalin anesthesia.) A portion of the hypertrophied middle turbinate of Mr. A., after being submerged in salt solution for about ten minutes, was transplanted into the tunnel. This piece of tissue, about a half inch long and a quarter of an inch in its other diameters, consisted of mucous membrane containing a sliver of bone. Bone placed in contact with the denuded septum, flap pressed down, apposition perfect, meatus very much narrowed. December 14, 1912, transplant still in situ, slight retraction of the mucosa at the line of incision, no pus. Patient complains of slight pain in the left eye. January 11, 1913, some granulating area at the line of incision. Probe introduced here gives the slight sensation of bare bone. A longitudinal incision over the entire length of the graft with the formation of a flap of septum mucous membrane was turned down, exposing the mucosa of the transplant, which appeared normal. February 11, 1913, transplant in excellent condition, closes up meatus to a very narrow slit.

Cases VI and VII.—Two attempts to close a septal perforation with transplanted mucous membrane. On April 22, 1912, I performed a submucous septal operation on Mr. C. S., aged —. During the operation the septum was torn at the site of the original incision. Thereupon I removed a small piece of mucous membrane from one of his turbinals and placed it into the perforation. This transplant failed to hold and came out in a few days. February 13, 1913, I again attempted to close the perforation, which was about a quarter of an inch in diameter. Cocain and adrenalin were applied, and a buttonhole incision made in front of the perforation. The mucous membrane surrounding this perforation was slightly elevated through this incision. A piece of mucous membrane somewhat larger than the perforation was taken from the hypertrophied middle turbinate of E. B., a healthy boy of thirteen years. It was preserved in salt solution for about thirty minutes before being transplanted, and was then placed under the elevated mucosa surrounding the perforation. Three days later the transplant was still in position and appeared somewhat dark in color. Eight days later the transplant was still in position except at a point occupying about a fourth of the circumference of the perforation. The transplant seemed viable and when rubbed became hyperemic. (Since the above was written the transplant has shriveled and come away.)

CONCLUSIONS.

If we analyze the results obtained in this rather limited series of experiments, we find that there are no especially technical difficulties in the transfer of intranasal tissue from one individual to another of the same species. Surface transplantation in animals will probably fail, owing to the difficulty in preventing infection. The submucous transplantation in animals should yield much better results, and the tissues seem capable of growing in their new environment. The experiments were not carried on long enough to determine what ultimately becomes of the transplanted bone, but it is practically established at the present time that transplanted bone is resorbed, that its framework serves as the scaffolding for new bone which is regenerated in its place.

In the clinical experiments, surface transplantation does not seem to offer very great prospects of success, although the grafts will adhere if the mucosa be slightly elevated to receive them. Submucous transplantation of nasal mucous membrane and underlying bone can be carried out with good prospects of the survival of the transplant without suppuration. It seems, however, that in the course of time the transplant tends to be absorbed. It may be possible to prevent this resorption by the removal of the roof of the tunnel in which the transplant has been placed. This procedure may stimulate the transplant to functionate and thus prevent absorption.

At any rate, facilities for attempting the transplantation of mucous membrane and nasal bone are abundant in every rhinologic clinic, and with due precautions as to the source of the tissues, these experiments may be advantageously continued.

BIBLIOGRAPHY.

1. Marx, G.: Zeitsch. f. Ohrenheilkunde, June, 1910, Bd. LXI, H. I.
2. Goldsmith, P. G.: Trans. Amer. Lar., Rhin. and Otol. Soc., 1910.
3. Kerr, I.: Woman's Med. Jour., January, 1911.
4. Von Eicken: Zeitsch. f. Ohrenheilkunde, Bd. LXI, H. 2, July, 1911.
5. Flatau: Ref. only, Zeitsch. f. Ohrenheilkunde, December, 1901, p. 302.
6. Axhausen, G.: Med. Klinik, November 19, 1911; also ref. J. A. M. A., December 23, 1911.
7. Lexer: Centralblt. f. Chir., No. 29, 1911.
8. Schoene: Centralblt. f. Chir., No. 29, 1911.

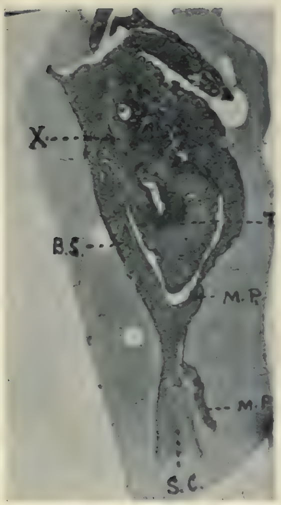


FIGURE I.

Section (magnification 4-1) through a transplant placed under the perichondrium and periosteum of the nasal septum of a dog. S. C., septal cartilage. B. S., bony septum. M. P., mucoperichondrium. M'. P'., same, elevated to form a pouch containing T, the transplant. At X the transplant is firmly united to the bony septum.

(Microphotograph by Dr. Chas. Goosmann.)

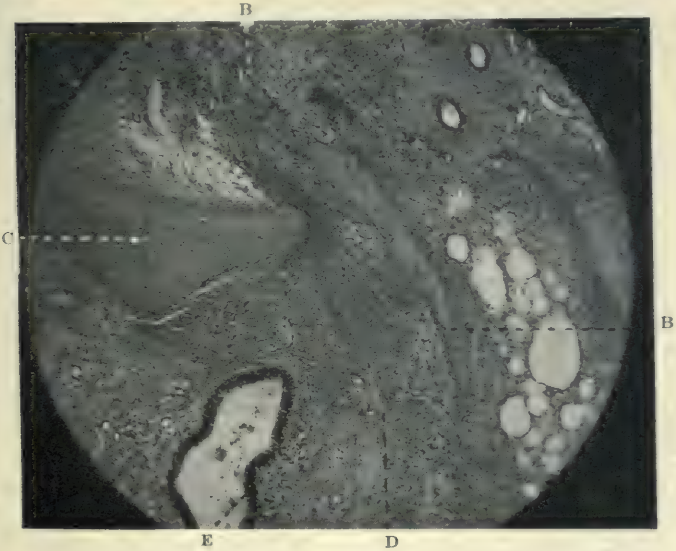


FIGURE II.

Higher magnification (100-1) of another portion of transplant illustrated in Figure I, showing intact condition of bone, B. B. Cartilage, C.; Connective tissue; D, and Glandular epithelium E. (Microphotograph by Dr. Chas. Goosmann.)

XV.

THE REPORT OF A CASE OF PARACOUSTIC VERTIGO AND NYSTAGMUS CURED BY OPERATION ON THE LABYRINTH.*

By John Randolph Page, M. D.,

New York.

A number of operations for the relief of vertigo have been performed on the nonsuppurative labyrinth by Milligan, Lake, Yearsley and others, and Yearsley in his report, "A Case of Severe Vertigo and Tinnitus; Destruction of the Labyrinth; Cure" (published 1908)[1] states that his was the ninth operation of its kind for the relief of distressing vertigo and tinnitus. "The first," he says, "was that of Milligan, the second that of Lake, who, however, antedated Milligan in publication." Since that time Mr. Lake, as he stated in his paper on vertigo before the New York Academy of Medicine last December,[2] has performed fourteen such operations. In this country the labyrinth operation for the relief of vertigo (so far as I have been able to learn) has never been performed before, at least there are apparently no available reports of any.

Last April a report of the history and examinations of this case was made before the Section on Otology at the New York Academy of Medicine.[3] Since that time his disturbing symptoms have been entirely relieved by an operation on his labyrinth. It will suffice to say now that the individual is a strong, healthy man, forty-four years of age, who since boyhood has been affected with a deafness and stuffiness in his right ear, which, until a year or more ago, he could considerably relieve by a movement of his lower jaw. He never remembers having had a discharge from either ear. Seven years ago he noticed that any exertion that caused increased heart action, such as lifting, running or even laughing heartily, made him stagger and lose his balance, and at the same time the objects seen by him seemed to move up and down with the pulsations of his heart.

---

*Read before the annual meeting of the American Laryngological, Rhinological and Otological Society, May 10th, 1913, Washington, D. C.

After November, 1910, certain sounds caused such disturbance of his equilibrium and, as he expressed it, of the objects before his eyes, that he would fall when he heard them, if he did not cling to something for support. He had to have the pitch of the whistles on his factory lowered, so that they would not affect him. Riveting machines and other loud sounds affected him in the same way.

Examination (October 6, 1911) revealed a markedly retracted drum membrane and a stenosed tube on the right side, with hearing for acoumeter and whisper, 2 inches and 18 inches, respectively. The left ear showed evidence of chronic middle ear catarrh, with hearing for acoumeter and whisper, 6 inches and 15 feet, respectively. The sound from a 256 fork placed on the vertex was definitely referred to the right or poorer ear. The low tone limit was practically normal in both ears (16 fork was indistinctly heard in the right, distinctly heard in the left ear). The upper tone limit in the right ear was reduced to 6.00 on the Edelmann-Galton whistle, and for the left ear, 2.00. Rinné was positive in both ears. Rotation tests with head in various positions revealed nothing unusual, and the caloric test was normal.

When the right ear was closed, sounds had no effect on his eyes or equilibrium. In the right ear, however, pipe sounds above a^3 (1702 double vibrations) produced marked disturbance until a^4 (3404 double vibrations) was reached. Sounds below and above this octave caused little or no disturbance, the most marked disturbance being caused by the middle notes of the octave. He stated that at the commencement of the sound the object he was looking at seemed to move downward and to the left, and remained in that position with a wavering movement until the sound ceased; it then returned to its original position. The eyes were easily seen to rise and fall when the sound was produced and stopped, and when the most effective sounds were produced his equilibrium was disturbed. At this examination the eyes seemed to move always in the plane of the right anterior vertical canal.

A month after this examination he was admitted to the

Manhattan Eye, Ear and Throat Hospital. A lumbar puncture was performed, and 20 cc. of normal cerebrospinal fluid was withdrawn, and at the same time, because of its marked retraction, an incision was made in the right membrana tympani. Intense headache, dizziness and nausea followed and persisted for the next forty-eight hours, and for two weeks the slightest exertion in the upright position caused severe headache and dizziness. A grateful feeling of clearness was temporarily afforded by the myringotomy, however. After three weeks the patient left the hospital, his vertigo much improved, and sounds that before would have made him fall had no effect upon him.

Three months after the lumbar puncture (the 9th of February) he reported that the staggering tendency had entirely gone, and he had no dizziness, but the right ear had become very sensitive to the sound of his own voice, and speaking above a low tone caused his eyes to jump. He could not whistle, call, or laugh without a jumping of the objects in front of him and a disturbance of his equilibrium. High tones no longer troubled him to the extent they formerly did, but lower pitched ones became so unbearable he had to raise the pitch of the whistles on his plant back to what they were before.

When he said E, P, T, Z, etc., or such a sentence as "Ease it off," objects in front of him moved downward and to the left, and his eyes could be seen to move upward and to the right in the plane of the right anterior vertical canal. If he whistled certain notes, a marked effect was produced, and if the sounds were prolonged, his equilibrium was upset. It was also found at this examination that when the note d^3 (1152 vibrations) was sounded on a pipe, the eyes moved straight upward, and he said the objects in front of him moved straight downward, giving him a sensation of marked disturbance. Above this pitch the disturbance was not so marked, but the objects seemed to move downward and to the right; and below this pitch they seemed to move downward and to the left.

Four months later (July 8th) the patient returned again for examination and reported that since his visit in February he had had a return of his vertigo—that is, staggering while walking; two attacks, so severe that he had to cling to a fence to keep from falling.

His chief complaint, however, was the effect of his own voice on his eyes. In speaking of this he pointed somewhat confusedly to his supraorbital region and the right side of his head. (It is well to say here that the man is an exceptionally strong character, and through force of will kept constantly at his work. A man less forceful and free from hysterical tendency would have succumbed long before.)

He had to acquire a very low tone of voice in conversation, otherwise the objects in front of him would vibrate up and down obliquely before him. Words like Ease, Peas, Disease, etc., would displace everything in front of him; and if he laughed heartily or said toot-toot-toot, things shifted so suddenly they caused him to lose his balance entirely. At each toot his eyes could be plainly seen to jump.

No less annoying was a pulsating sound like a drum in his right ear, and although he could not remember the time when he was not able to count his pulse by hearing it in his right ear, the present sound was a new development, and it became more marked after mental or physical labor. His only relief when it became unbearable was to lie down for fifteen or thirty minutes; the eye symptoms then subsided entirely and the pulsation ceased except for a slight sound. In using the large and small pipes of the Bezold series, it was found that d^3 (1152 vibrations) on the small pipe caused objects to move straight downward, as was the case at the examinations in February, with marked disturbance (wide movement). The patient could not stand the sound for any length of time, and if it was tooted his equilibrium was upset. a^4 (3404 vibrations) caused objects to move more laterally at about an angle of 45° to the right and downward—the eyes in the opposite direction. Above this point no disturbance was obtained. The lowest note on the large pipe e^2 (640 vibrations) gave marked disturbance downward and to the left. His response to some of the intermediate notes was at times contradictory, owing, he said, to the confusion from the sounds, which caused him to fail to notice whether the movement was first up and then down or vice versa.

I insert here my record of the effects produced by the different pipe notes, not that I am able to interpret anything of value from it, but simply that it may be recorded in case it may excite the interest of someone with a theory to support:

d^3 (1152 vibrations) on small pipe: Objects move straight downward, as before (in February), with marked disturbance.

e^3 (1280 vibrations): Even more disturbance, but downward and slightly to the right.

f^3 (1363.2 vibrations): Same. with less disturbance.

g^3 (1536 vibrations): Practically no disturbance and slight movement.

a^4 (3404.8 vibrations): Objects move more laterally at about 45° to the right, eyes move in the opposite direction; above this point of a^4 no disturbance of eyes is obtained.

g^4 (3072 vibrations): Objects move upward and slightly to the right.

f^4 (2726.4 vibrations): Objects move upward and beginning to curve towards the left with marked disturbance.

c^4 (2048 vibrations): Obects move upward and a little to the left.

a^3 (1702.4 vibrations): Objects move upward and a little to the left.

g^3 (1536 vibrations): About the same.

e^3 (1280 vibrations): Almost gone.

d^3 (1152 vibrations): Getting worse again.

c^3 (1024 vibrations): Bad; upward and to the left.

f^3 (1363.2 vibrations): Upward and to the left.

a^2 (851.2 vibrations): Straight down.

g^2 (768 vibrations): Marked disturbance. up and down to the left.

f^2 (681.6 vibrations): Marked disturbance, down to left.

e^2 (640 vibrations) lowest pitch in pipe: Gives marked disturbance downward and to the left.

This examination was confusing and fatiguing for the patient, and he himself remarked that he could not observe accurately because of his dizziness and disturbance. This probably accounts for the contradictory answers.

As the patient's disturbing symptoms seemed to grow worse rather than better, operation for the destruction of his labyrinth was at last resorted to. A pneumatic mastoid with normal looking cells was opened, and there was no evidence in either the mastoid or the middle ear of necrosis of any kind.

In attacking the labyrinth the horizontal and anterior vertical canals were opened and traced forward until their ampullæ were reached. These were removed, and the canals were then followed backward and the vestibule was entered posteriorly through the solid angle. The stapes was removed, and, because of the acoustic disturbance, it was thought best while at it to open the cochlear also, so the promontory was then cut away.

An instrument devised by Dr. Kerrison, which easily shaves the hardest bone, enables one to avoid the use of chisels and the accidents which sometimes follow their use in this operation. Particularly those accidents referred to by Dr. Richards:[4] namely, the tendency of the external canal to crack through definite lines of cleavage into the fallopian canal and cause injury to the nerve, and the modiolus to crack at its base and open the internal auditory meatus with an escape of cerebrospinal fluid.

Judging from appearance only, it might have been a normal labyrinth that was being operated on. The eustachian tube was curetted thoroughly down through its isthmus with Yankauer's instrument designed for that purpose, but there was no antiseptic treatment of the wound, as I had not then heard Mr. Lake advise it as a wise precaution. The wound was washed with normal salt solution and packed with plain gauze, a little iodoform gauze being placed next the labyrinth itself. Ten days later a meatus and flap were cut and the incision was closed behind. Three weeks later the cavity was skin grafted. There was no exposure of the facial nerve and no paralysis.

For several days after the operation the patient lay on his sound side, hunched up with his eyes closed. Nausea and vomiting persisted for four days.

At an examination on September 9, 1912 (five and a half weeks after the operation), he stated that after the fourth day following the operation the disturbance of objects was not as great with his left eye open as it was with both eyes open, and he appeared to have less control over the right eye than over the left; the disturbance was more marked when objects were seen with the right than with the left eye. He had diplopia with both eyes open, which persisted for a week. Even at that time (September 9th), six weeks after the operation, he had diplopia when he first opened his eyes in the morning; a disturbance he had never had before the operation. He stated that after the labyrinth operation, despite the intense nausea and vomiting, he noticed that objects moved from "high left to low right;" that is, they moved from left to right downward at an angle of twenty degrees. This persisted for a week and gradually diminished until there was no movement of objects except when he first awoke in the morning. Six weeks after the operation, on waking in the morning, objects still seemed to move slowly in this direction, but after a few minutes he would be able to overcome the illusion.

Never after the operation was he disturbed by any sounds in his ears. In this he differs from Dr. Richards' case, to be mentioned later, of the man who suffered marked disturbance from sound after his labyrinth had been removed. An attack of vomiting, or any exertion, during the first week or two after the operation, would cause a gradually increasing sound, "like the passing of a subway train," in his operated ear; it occurred first faintly, and then became louder and louder like a very heavy storm, lasting a short time and then gradually subsiding. These noises became less and less frequent, but they were occasionally noticed for three weeks after the operation. Never since the operation has sound had any effect on his equilibrium, or on the movement of objects, or on his eyes themselves; nor has increased heart action caused any disturbance whatsoever. He can whistle and toot-toot-toot with great satisfaction and without any disturbance to him of objects or of his equilibrium.

On the ninth day after the operation the patient was allowed to sit up in bed, because he desired to do so, and thought he could. The movement, however, caused a return of the dizziness, and after a few minutes he was glad to lie down again. On the eleventh day he was allowed to stand on his feet, but he would have fallen to the floor towards the operated side had he not been supported. He persisted in getting up, however, and three days later was able to walk the length of the hall unassisted, though he was very unsteady, and had a tendency to fall towards his operated side. Turning to the left at first caused a little disturbance, but this soon disappeared. On turning to the right, however (to the operated side), marked disturbance was produced, and even a movement of his eyes to the right at the examination, six weeks after the operation (September 9th), was accompanied by a movement of all the objects to the right. This movement of objects became less every day. The patient was then able to walk without falling, with his eyes closed. He had practiced whirling, turning and walking backwards until he had excellent control of himself, although all signs of disturbance had not entirely disappeared. There was still some nystagmus on looking to the left, practically none on looking to the right. On rotation to the left ten times, after-nystagmus was slight and lasted only twelve seconds. On rotation to the right, after-nystagmus lasted only fifteen seconds, but he complained of considerable disturbance and felt a little sea-sick, although the seasickness was only momentary.

An examination made November 11, 1912 (over four months after the operation), showed the cavity well dermatized except for small granulations which persisted at the opening into the vestibule, posteriorly. From this region there seemed to be a slight leakage of clear fluid, and when it was irritated with an applicator, some disturbance of equilibrium and objects was remarked by him.

The patient was greatly improved, and neither exertion nor sound gave him any further trouble. He had no disturbance of equilibrium except when he turned quickly to the right. He was still, however, unable to walk with certainty with his eyes closed, or in the dark.

A letter received on the 11th of January, 1913, from Dr. A. P. Butt, his physician, stated that the ear was healed; and one from the patient himself, about the same time, informed us that he feels splendidly all the time. In answer to questions, he says he is not yet able to turn to the right quickly without disturbance, and that turning his head and eyes suddenly to the right causes him to sway. He still experiences difficulty in walking at night in the dark, or with his eyes closed, though the difficulty is becoming less. There is no movement of objects after exertion, or pounding in the ear, as there was before the operation. There is no disturbance of equilibrium due to sound, either external or from his own voice. He still has a slight hissing tinnitus, which he had before the operation, a point worth noting in regard to operation on the labyrinth for persistent tinnitus.

With the exception of Dr. Richards' case, and Mr. Lake's two cases, one of the man whose vertigo was brought on by exposure to a bright light and loud noises,[2] and the other whose attack followed his listening to a band,[5] in none of the many cases of vertigo reported have I been able to find sound mentioned as a casual factor, though Dench says:[6] "It should be borne in mind that when any portion of the auditory apparatus, either of the middle ear, the labyrinth, or the nerve trunk, is in a pathologic condition, any slight stimulus—particularly an increase in the blood pressure—may be sufficient to bring on an attack of vertigo."

The cause is frequently found to be due to derangement of the sound transmitting apparatus or to changes in blood pressure; but, as I said before, with the exceptions mentioned, I have found no case where sound itself was the exciting cause of the vertigo.

In regard to blood pressure, I recall here that Mr. Lake recently in his remarks after the presentation of his paper before the Academy of Medicine in New York, speaking of its influence on vertigo, seemed of the opinion that the virtue claimed by many for lumbar puncture was due solely to its effect on the blood pressure, and that a more lasting benefit could be obtained with depletives and drugs in such cases than with lumbar puncture itself. In this he does not agree with many who have for some time advocated lumbar puncture without very definite reasons for expecting permanent relief from it, and more recently with Bárány,[7] who claims for it marked benefit, and often permanent relief in certain cases where the vertigo and "Symptomen-komplex" are due to adhesions of the pia in the region of the cerebellopontine angle which are broken up by the sudden withdrawal of the cerebrospinal fluid.

Blake[8] calls attention to the condition of the sound transmitting apparatus as playing an important part in the causation of vertigo, but mentions nothing about the effect of sounds on it.

A comparison of this case with the one Dr. Richards[4] mentions in his "Treatment of Purulent Affection of the Labyrinth," etc., etc., forms an interesting paradox. In his case, disturbance of equilibrium was caused by certain sounds after the removal of the labyrinth, and in this case the opposite was true, in that the disturbance caused by certain sounds was cured by the removal of the labyrinth.

Dr. Richards tells me that he removed not only the labyrinth, but almost the whole of the petrous pyramid for suppurative labyrinthitis in his case (a young musician), and that the young man while convalescing seated himself at the piano to play, but when he struck the keys he was knocked off the stool by the sound, and for months afterwards, even after the cavity had healed, musical sounds from the piano disturbed his equilibrium. He also says that while dressing this case, when he touched the stump of the nerve, or the soft tissue about it in the region of the internal auditory meatus, which he had opened, the patient never failed to remark a noise in that ear, but when the bone near that region was touched with a probe no sound was heard.

Following the mention of this case in his article, he says: "It would appear that we receive through the auditory apparatus certain physical impressions which, when transmitted to higher centers, act as supporting influences to the preservation of station; that these, when not balanced by similar impressions acting upon the other ear cause, so to speak, an auditory imbalance; that the disturbance is due to the lateralization chiefly to one side of those impressions which give rise to the subjective phenomenon of sound is questionable, inasmuch as the patient, who was totally deaf before operation, experienced then no such disturbance under similar conditions." He continues, saying: "The phenomenon is suggestive of the great variety of factors which may act through the medium of the auditory apparatus and contribute to the preservation of that condition which we speak of as the state of equilibrium—it gives us an insight into the intricacy of the function of the auditory apparatus."

The disturbing, jangling effect of musical instruments, pianos particularly, on ears recovering from acute middle ear suppuration or some other disturbance of the conducting apparatus, is a familiar one, and although the effect is not usually so great as to cause loss of balance, it not infrequently causes a sensation closely akin to it.

The association of the acoustic with the static labyrinth in the subject of this report is interesting, and it along with the views of Richards, as expressed above, might be considered by some to support v. Cyon's theory, an expression of which I quote from Dr. Fridenberg's[9] review of v. Cyon's theory of the labyrinth: "v. Cyon proceeds to develop his own theory, which brings the elements of tone and sound perception into relation with the innervational control and regulation, both as to intensity, succession, and duration, of the entire muscular apparatus of the body, especially of the eye; with the rhythmic coordination of speech and with the psychic control of function. This coordination depends on concepts of number which can only originate in the cochlea, and is associated with concepts of direction, dependent physiologically on stimulation, continuous or intermittent, of the semicircular canals."

Later, in his criticism of v. Cyon's theory, he says: "Biologically (too), auditory sensations can hardly be made use of as a basis for an adequate stimulus of the semicircular canals, as these sensations have not yet appeared in lower animals which are capable of perfect orientation."

In the case just reported, it would seem that when the auditory apparatus was stimulated by sounds of a certain number of vibrations, a definite movement of the eyes took place in the plane of a certain semicircular canal in the diseased ear. So, according to Flouren's law, there was caused by the sound vibrations just as definite a movement of endolymph in the ampulla of that canal as there was by the pump acting directly on the canal in Ewald's experiment. But pulsations from the heart also at times caused the same disturbance, so the commotion in the ampulla cannot reasonably be associated with the effect on the auditory apparatus alone. It would seem, rather, that the effect on the ampulla was coincident with but separately caused by the (sound) vibrations, which transmitted through the cochlea were interpreted as sound.

With this conception one can still maintain the theory of the distinct separation functionally of the auditory from the static labyrinth, and receive the support of the biologists and that of long general acceptance as well. One can still support this theory and realize that the localization of sounds may play a part in orientation, just as localization of objects by means of the eyes do, and the muscle and tactile senses do, but that does not mean that one has to accept the view that such a close association exists between the two, static and auditory, that sound through irritation of the cochlea influences the endolymph in the ampulla, for it is only when the contents of the ampulla are abnormally sensitive or irritable that the same quality of vibration affects them both.

BIBLIOGRAPHY.

1. Yearsley, Macleod: A Case of Severe Vertigo and Tinnitus; Destruction of the Labyrinth; Cure. The Lancet, September 19, 1908.
2. Lake, Richard: Aural Vertigo (Non-Suppurative): A Clinical and Therapeutical Study. The Lancet, December 14, 1912.
3. Page, John R.: A Case of Paracoustic Vertigo and Nystagmus (Preliminary Report). Annals of Otology, Rhinology and Laryngology, September, 1912.
4. Richards, John D.: Treatment of Purulent Affection of the Labyrinth Consecutive to Disease of the Middle Ear. Annals of Otology, Rhinology and Laryngology, September, 1907.
5. Lake, Richard: Ten Cases of Operation for Meniere's Disease (Aural Vertigo). The Lancet, June 10, 1911.
6. Dench, E. B.: Vertigo from the Standpoint of the General Practitioner and Otologist. New York Medical Journal, January 6, 1912.
7. Der Baranysche Symptomen-komplex, seine Diagnose und Therapie, Aetiologie und Prognose. Verhand. d. Deutsch. Otol. Ges., 1912.
8. Blake, Clarence J.: Vertigo of Aural Causation. Boston Medical and Surgical Journal, October 6, 1905.
9. Fridenberg, Percy: Space and Time as Aural Concepts: A Review of v. Cyon's Theory of the Labyrinth. Laryngoscope, St. Louis, October, 1909.

XVI.

ATROPHIC RHINITIS WITH OZENA—ITS ETIOLOGY AND SURGICAL TREATMENT.*

By Francis Patten Emerson, M. D.,

Boston.

INTRODUCTION.

From the discussion of atrophic rhinitis with ozena, as found in the literature, it is evident that special investigators are particularly interested in two methods of procedure in the study of the cause and treatment of this disease. First, in an attempt to isolate a specific organism and establish a method of treatment based on such findings, and second, in looking upon the symptom complex as due to pus producing organisms; causing multiple foci that are amenable to the recognized surgical principles of drainage and cleanliness.

The difficulties encountered by an investigation along the first procedure are due to the results of decomposition and secondary infection, which give rise to an innumerable bacterial flora and interferes with an attempt to determine which particular one is the active organism in the disease.

Renewed interest has been awakened of late by the work of Abel and his experiments with the bacillus mucosus ozenæ. This painstaking work has led others to attempt to substantiate his claims. An excellent article, among others, appeared last year in this country, by Cobb and Nagle. This phase of the subject has already in previous years engaged the attention of members of the profession, notably, Lowenberg, Pesand, Gradenigo, Dreyfuss, Loeb, Ambi, Brindell, Gruenwald, Hajek, Marano, Abel, and others, but without any definite results.

In the second method of procedure, where the symptom complex is considered as due to an attenuated infection with multiple foci, secondary to a pansinusitis, this would suggest that the original infection is not followed by an essential or primary atrophic process acting as an intermediate step, but that the sinuses are invaded directly by a pus producing organism. While it is conceivable that fetor and crusting might be produced by the breaking down of soft tissue, bone necrosis and defective drainage, with the resulting secondary infection, yet we know that certain pus producing organisms by themselves are capable of causing a thin, others a thick, secretion, and still others may cause fetor and crusting. In this sense a specific organism might be necessary to produce ozena, but not necessarily have, as its primary manifestation, an atrophic tissue change. If we accept the latter view, which the writer believes is true, we can with advantage continue to search for a specific organism and at the same time perfect our surgical technic, because purulent retention within nonelastic walls requires free drainage, as a prerequisite to cure, whatever the infective agent might be.

---

*Read before the Nineteenth Annual Meeting of the American Laryngological, Rhinological and Otological Society, held at Washington, D. C., May 8, 9 and 10, 1913.

THEORIES.

From the earliest medical records to the present time, many theories have been advanced as to the etiology of ozena, but none of them have had a considerable following among men of eminence. For a review of this literature you are referred to the excellent article by Sendziak in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY for 1907.

That tuberculosis and syphilis cause a certain number of cases is conceded, but the researches of Sobernheim, Eisenlohr and Alexander by the aid of the Wassermann test showed that syphilis, the disease most suspected as a cause of ozena, was responsible for relatively only a small percentage.

Bosworth's theory of a preceding purulent rhinitis, with no sinus involvement, as a cause of ozena in later life, has been questioned by the researches of Pearce at the Boston City Hospital, and of Pearce and Winne at the Albany Bender Laboratory, who showed conclusively that the sinuses are often invaded by a pus producing organism in the course of the infectious diseases as early as the second year. Is it not also the experience of all rhinologists, with their present better knowledge of sinus infections, that a primary purulent rhinitis is a thing of the past? It is always secondary to some focus of infection in the epipharynx or hollow spaces.

Theories based on the shape of the face as being a cause are less often referred to today, with our more definite pathology on which to base a diagnosis.

The Herd theory of a sinus disease as a cause of ozena, has had such able exponents as Gruenwald, Hajek, Loehnberg, Michel, and not a few among the members of this society. Although this theory has not been generally accepted, yet the writer brings this phase of a much discussed subject before you today again, because of his belief that the rapid advance in our knowledge of and ability to cure sinus disease, has opened new possibilities in the study of the etiology and cure of ozena, even granting a specific organism.

In the past, when treating these focal troubles intranasally, even if we agreed as to its etiology, yet our knowledge of the surgical technic of sinus disease has advanced sufficiently to make it possible to adopt a common method of common exenteration of involved cells. This would in turn lead us to some definite agreement for or against a focal infection, as being the chief underlying cause of this disgusting affection.

PERSONAL WORK.

It is for this purpose that I venture to offer the results of the following surgical procedure, after briefly stating the grounds which, in my opinion, implicate the accessory sinuses, without there being a true atrophic rhinitis.

First, the writer has never seen a case of a socalled atrophic rhinitis that was not relieved by thorough drainage, instead of being made worse, as would be the case if trauma and increased air space were added to a genuine atrophic mucosa.

Second, the crust formation and fetor, when unilateral, are always on the wide side, where they have apparently been preceded by a compensatory hypertrophy of the second turbinate, due to the deviated septum. This enlarged turbinate would in turn interfere with the proper drainage of a subsequent sinus infection.

Third, the discharge of pus over a mucous surface in a long standing case is, in the writer's experience, always accompanied by a glazed appearance of the mucosa, dryness and an exudate, a low grade catarrhal process, and not a true atrophic process. That this is not a true atrophy is shown by the return of function of the mucous surface with the establishment of free drainage and the cure of the underlying focal infection. This is often seen on the posterior wall of the pharynx in cases of chronic sinusitis of the sphenoid cavity or posterior ethmoid cells.

Fourth, radical surgery, with the object of establishing free drainage, lessens the tendency to crust formation, instead of increasing it. The lessening of crust formation is in proportion to the thoroughness with which the affected cells are drained. This is followed by a purulent or mucopurulent discharge, until the diseased surface is made healthy by topical applications and cleanliness. Nowhere in the body does nature tolerate so much abuse as in the nasal chambers, if we provide for free exit of the secretions.

Fifth, ethmoid disease, the writer believes to be the most constant of the infectious processes in the nasal chambers. As a suppurative ethmoiditis it is easily recognized. On the other hand, there are inflammatory processes of the ethmoid cells which are less active, or where the drainage is better, in which case they are characterized by serous infiltrations, dilatation of the capillaries, with the mucous membrane dry and red; and if seen after the process has been going on a certain length of time, there is found covering the mucous surface an exudate which is made up of dead epithelium, pus, mucus and blood. These seem to be the steps and the macroscopic appearances clinically of a certain type of catarrhal ethmoiditis. This type of an inflammatory process, where it fails to undergo resolution, passes into a chronic condition, which the writer has repeatedly noticed, was spoken of by many clinicians as atrophic rhinitis. Since these cases of atrophic rhinitis get well under improved drainage, the question would, therefore, arise, whether the clinical term of "atrophic rhinitis" is a misnomer, or the pathologic understanding of atrophy is incorrect. To the writer it is inconceivable that a true atrophy with destruction of the glandular elements and an increase in the connective tissue stroma should resume its function clinically, as is the case of the mucous membranes of the nasal chambers where a free exit for the secretions is provided. Such cases untreated may continue indefinitely, but when the ostia become obstructed by inflammatory changes, and drainage is interfered with, a subsequent active infection may take place, resulting in a suppurative ethmoiditis.

Sixth, the general systemic symptoms, such as pallor, loss of weight, muscular weakness, gastric and other symptoms, are a sequel rather than a predisposing cause of the local condition. These symptoms are found to improve with diminished absorption as the local condition is relieved.

Seventh, the writer's cases of ozena have shown the morbid process more uniformly to be in the ethmoid and sphenoid sinuses. Next in frequency have been the ethmoid, sphenoid and frontal, and last the ethmoid, sphenoid and antrum.

The preceding observations were further strengthened by the classical work of Gruenwald on nasal suppuration, and the report of cases by Michel, Loehnberg, Hajek, Mayer, Beck, Theissen, and others. Although in the beginning of this work the cases were tedious and necessarily difficult to cure, owing to the haphazard way individual cells were attacked at that time and their proneness to relapse with reinfection, yet the results during the last four years have grown increasingly more satisfactory with added experience and a better technic. I wish to emphasize right here that the successful results in the treatment of these cases of socalled atrophic rhinitis with ozena depends absolutely upon the careful after-treatment, which is fully as important as the thorough surgical exenteration. Regular and painstaking care must be exercised until all the affected surface is brought into a healthy condition; and even then for a long time we have a membrane with low vitality, easily reinfected and functionally disturbed.

GENERAL DESCRIPTION OF CASES.

The clinical cases fall into two general classes: one the typical cases of Fränkel, with crusting, fetor, anosmia, necrosis and sloughing of the soft tissue, and the second the atypical cases in which fetor and crusting may be absent, and where the diagnosis of attenuated sinus infection might be more an open question. Contrary to the established facts in the treatment of disease, the older the stage of this disease the easier it is to cure. This is in perfect harmony with the sinus theory of the etiology of ozena, owing to the fact that in the third stage, where the infection is less active, the ostia open and the drainage more or less perfect, due to the necrosis and the sloughing of the soft tissue, there is left a smooth bony surface, and therefore the cure is relatively easy.

In the second stage, although earlier in the disease, with the mucous membrane boggy, drainage blocked, and the entire surface covered with a thick, firm crust, the problem is increasingly difficult.

The element of fetor has not seemed to the writer to indicate that a case would present any additional obstacle to healing, for the reason that the odor may be due to a particular organism, not necessarily more virulent or more intractable to overcome than those with little or no odor. In fact, the only serious problem is an anatomic one. In those cases where the cells are developed in the floor of the frontal sinus or out over the orbit, it is obvious that drainage must be obtained by extranasal methods. If we assume that drainage is essential, then we must admit that a certain percent of cases of ozena, presenting these anatomic peculiarities, cannot be cured by any intranasal procedures, whether these are along medical or surgical lines. These variations in development have been pointed out by Shambaugh in an excellent article entitled: "The Construction of the Ethmoid Labyrinth," in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Volume 16, 1907.

We approach our surgical procedure then with the understanding that—

First, for anatomic reasons, all cases cannot be cured intranasally.

Second, that when functionally cured, the previously infected surface is prone to relapse, due to reinfection.

Third, that only constant attention to drainage, the removal of polypi, topical applications and cleanliness, following radical exenteration, will avail in any case.

Given these conditions, the writer believes that as large a percentage of cases of ozena can be brought to a successful issue as we could expect to cure intranasally, cases of undoubted pansinusitis.

SURGICAL TECHNIC.

The first step consists in anesthetizing the patient in a reclining position. He is then placed in a chair and the nasal mucosa is adrenalized. The sphenoid opening is next located with a probe, and if not easily accessible, part of the second turbinate is removed, in order that we may take away enough of the anterior sphenoidal wall, so that the enlarged sphenoid opening can easily be recognized when the landmarks have been removed. Next the method of Mosher is employed to systematically exenterate under direct vision the ethmoid labyrinth. With the head tipped back the agger nasi cell is located a little above and in front of the attachment of the second turbinal, corresponding to the rounded eminence on the outer nasal wall. This is found with a curette, by first feeling for the hard surface of the superior maxillary process and then working toward its posterior margin until you break through the thin outer wall of the cell, which corresponds to a large cavity lying at the floor of the nasofrontal duct. It is now important to bring the head forward so that it is at right angles to the body. The curette is then turned so that it faces the nares, away from the orbit. With a lateral to-and-fro motion the inner cell walls and partitions can with a little practice be easily broken down back to the posterior ethmoid cell by the sense of touch. The point here which the writer wishes to emphasize is that the curette is not to be used to remove the lining membrane, but as an instrument to break down the bony partitions and nasal wall of the cells.

This leaves the second turbinate turned down and attached by a narrow base which can be wholly removed by the nasal scissors or forceps in a few minutes. We next pack the operative field with strips of gauze wet in a solution of adrenalin 1/3000. We can now work on the opposite side, where our original operative procedure is duplicated and the field tamponed. The gauze in the first side is then removed, the enlarged sphenoidal opening sought, and its remaining anterior wall is then removed up to the posterior ethmoid cell. The field is then gone over by introducing the little finger into the nares in order that we may detect any irregular bony projections or margins of cells that may interfere with drainage. All granulations and polypi should be removed without destroying the entire mucous lining. The patient is then returned to bed without any nasal packing.

AFTER-TREATMENT.

First, mechanically remove all crusts gently, to avoid bleeding. If the drainage is good, after the first week or ten days these will cease to form and the discharge will have become mucopurulent.

Second, after the first week remove all polypi that may appear. These will tend to return for several weeks.

Third, use the Bier suction apparatus, after first washing out the sphenoid cavity with a canula. Watch especially the sphenoid opening at its margin, where it tends to granulate in rapidly and obstruct drainage, as pointed out by Hajek. Touch the margin with silver nitrate or trichloracetic acid until it remains permanently open as the result of cicatrization.

Fourth, make the surface dry by gentle pressure, and after the first week apply a 4 to 8 per cent solution of nitrate of silver, especially to the sphenoid cavity itself and over all unhealthy areas wherever found.

Fifth, if the patients live at a distance, allow an alkaline wash to be sniffed twice daily, from the hand or a tablespoon. The surplus water should then be blown from the nares without taking hold of the nose, on account of the danger of back pressure in the eustachian tubes.

Sixth, do not allow a water spray of any kind to be used. For four weeks the patient should be seen two or three times a week; then for four weeks more, once a week, and after that at irregular intervals until discharged. Remember to caution the patient that an acute infection will bring on a relapse which should be attended to at once. This usually means only a few treatments after the acute infection subsides.

GENERAL SUMMARY.

The interest of the profession in the etiology of ozena seems, from the literature, to be centered upon two methods of procedure. First, in an attempt to isolate a specific organism; and second, in looking upon the symptom complex as due to pus producing organisms causing multiple foci.

The difficulty in isolating a specific organism arises from the innumerable bacterial flora resulting from decomposition and secondary infection.

The secondary procedure does not admit an essential or primary atrophy as an intermediate step in the disease.

Theoretically, a specific pus producing organism might cause fetor and crusting without a true atrophic process.

That tuberculosis and syphilis are the underlying cause of ozena, except in a small per cent of cases, is not probable.

Bosworth's theory of a preceding purulent rhinitis in infancy without a sinus infection is questioned. The postmortem reports of Pearce and Winne show sinus involvement as early as the second year in the course of the infectious diseases.

More definite pathology has led to the abandonment of many former etiologic theories. The rapid advance in our knowledge of and ability to cure accessory sinus disease, makes it now possible to adopt a more uniform surgical technic for their exenteration. This should show more definitely the relationship of sinus infection to ozena. What seems to be an atrophic process is always improved by drainage.

A sinusitis frequently develops in the following manner: A septal deviation is followed by a compensatory hypertrophy of the second turbinate, with a resulting catarrhal ethmoiditis. This hypertrophy and catarrhal inflammation so interfere with drainage that a subsequent infection by a pus producing organism makes a sinusitis probable.

A chronic catarrhal inflammation accompanied by an exudate is frequently spoken of as an atrophic process. The return of function with improved drainage shows that it is not a true atrophy. Radical surgery, by establishing better drainage, lessens the tendency to crust formation. Is atrophic rhinitis then a misnomer?

The general systemic symptoms are secondary. The writer's cases have shown the morbid process more often to be in the ethmoid and sphenoid sinuses; next often in the ethmoid, sphenoid and frontal, and last, the ethmoid, sphenoid and antrum.

There are two types of clinical cases, one the typical cases of Fränkel, and the other the atypical cases, with fetor and crusting. The atypical cases are less open to question in regard to sinus involvement. The third stage of the disease is more easy to cure. The after-treatment is as important as the surgical exenteration. Fetor does not indicate a more intractable case. The serious obstacle to cure arises from an abnormality in the development of the bony cells. When such abnormal cells are present, certain cases of ozena are incurable by any intranasal method. All cases are easily reinfected, but respond readily to local applications, if the drainage is good.

CLINICAL CASES.

Case 1.—Mrs. H. C., aged 39 years, born in Norway, married.

Diagnosis.—Bilateral atrophic rhinitis with chronic ethmoiditis and sphenoiditis and a traumatic perforation of the septum.

Operation.—Radical exenteration of the ethmoid labyrinth and sphenoid sinus on the right side, leaving the left as a check.

History.—Family history negative. Pain general over the head and face for five years. Profuse discharge from the nose for two years. Has had several previous nasal operations.

Examination.—The entire nasal septum is destroyed to the ethmoid plate, leaving a large central perforation. Both inferior turbinates are shrunken and covered with crusts. Part of the middle turbinate remains. There is a large opening on either side of the cribriform plate. An X-ray shows double ethmoid clouding. The left frontal region is unsatisfactory. Thick scabs are general over both sides, and underneath there is a fetid purulent secretion.

Operation.—September 16, 1911. Dr. Emerson. Owing to previous operations, the landmarks are destroyed. There is some difficulty in locating the agger nasi cell. The ethmoid region between the posterior ethmoid cell and the anterior is one solid bony mass, with the wall too thick to penetrate safely. The anterior and posterior region was thoroughly opened and the remains of the turbinate removed.

Operation.—February 12, 1912. Dr. Emerson. Exenteration of the left ethmoid labyrinth and sphenoid under ether. Polypi in left posterior ethmoid cell removed. Partial remains of the right anterior sphenoidal wall removed.

Result, March 23d.—The right side has been free from crusts and odor for two weeks. The left side has some free pus, especially in the region of the nasofrontal duct and cribriform plate. Some headache on the left side.

Note.—This case was followed at the writer's office for some weeks until he was convinced that there was trouble in the left frontal sinus. This was uncertain from the X-ray plate. A Killian operation was then performed, which showed that the ethmoid cells extended out of the orbit and that there was a large cell in the crista galli. The frontal sinus was full of granulations, and considerable pus was seen coming from the cell in the crista galli as well as from the sinus. Such a case could not be drained intranasally.

Result.—The right nose has been clear for months. The left has a few drops of clear pus coming from the frontal sinus.

Case 2.—A. H., born in Ireland, aged 26 years, domestic, single.

Diagnosis.—Bilateral atrophic rhinitis with chronic ethmoiditis and sphenoiditis.

History.—Family history, one brother has bilateral ethmoiditis. Mother and one sister has goiter. No specific history. Measles at nine years. Frequent colds and discharge from the nose for five years. Pleurisy four years ago for a week. Occasional headaches. Odor from the nose has made it difficult for the patient to retain her position as a domestic. She is not strong and tires easily.

Examination.—Patient poorly nourished. Cheeks flushed and tongue coated. Alæ flattened. Septum deflected to the left. Whole outer nasal wall on both sides covered with a thick crust that can be removed with nasal forceps en masse. Under this a bleeding surface and free stinking pus. Report from the Boston Consumptives' Home showed lungs and sputum negative.

Operation.—Exenteration of ethmoid cells and anterior sphenoidal walls.

Result.—Patient was treated at intervals for a year, as the ethmoid region was exenterated at intervals under local anesthesia. For two years the patient has been entirely well, with no odor or dryness, and the mucous membrane is clean. The sphenoidal openings are large and clean. She has gained twenty pounds and has used no wash for a year.

Note.—During the last year the patient has been treated only twice, following an acute infection. This patient was one of the most marked cases of ozena, and she now shows a perfectly clean nose without the use of any wash.

Case 3.—A. E., aged 32 years, born in Russia, grocer, married.

Diagnosis.—Bilateral atrophic rhinitis with chronic ethmoiditis and sphenoiditis.

Operation.—Radical exenteration of the ethmoid cells and anterior wall of the sphenoid under ether.

History.—Family history negative. Patient referred for an examination for a possible focal infection as a cause of his endocarditis. There is crusting and discharge from the nose with loss of smell. Duration, seven years. He has been to several hospitals, and was told that his disease was incurable. He is weak and finds it difficult to do his work.

Examination.—Crusting over both outer walls of the nares, with very offensive discharge underneath. The second turbinates are shrunken. A large quantity of free pus is seen in the right middle meatus. The bridge of the nose is somewhat depressed. The septum is deflected to the left at the upper third. The right sphenoid cavity is very large, the left nearly obliterated.

Result.—The tendency to crusting stopped in two weeks. There was persistent discharge from the region of the right sphenoid until it was discovered that there was a partition in the sinus with a narrow vertical ostia close to the septum, leading into the main cavity, which had not been opened. The partition was removed and the cavity irrigated; then treated with silver nitrate 8 per cent. Further trouble was experienced by the tendency of the margin of the anterior wall to fill in with granulations. Finally, by the use of silver nitrate and trichloracetic acid to the margin, this was overcome and the cavity remained permanently open and the discharge ceased.

Case 4.—H. H., shoemaker, married, born in Russia, aged 31 years.

Diagnosis.—Bilateral ethmoiditis and sphenoiditis with ozena. L. O. M. S. C.

History.—Severe headaches for eight years. (The writer had performed a radical mastoid on the left side, and in doing the dressings the foul, sour odor from the nose was very marked.

Examination.—Both nares contain a considerable amount of foul pus with crusting. The middle turbinates are boggy and myxomatous at their anterior ends. Anosmia complete.

October 18, 1912, both ethmoid labyrinths were exenterated from before backward, under ether. Both anterior walls of the sphenoid were removed and the two cavities connected.

October 20, 1912, nose clean and no perception of odor.

October 31, 1912, patient discharged from the hospital and treatment continued at the writer's office.

November 10, 1912, discharged.

Result.—The patient moved to Michigan and was treated there four times, as the nose was not completely healed.

April 22, 1913, examination by the writer showed the nares free from crusts or discharge. Both sphenoid cavities are widely opened.

Case 5.—A. M., born in Nova Scotia, aged 44 years, married, furniture mover.

Diagnosis.—Bilateral atrophic rhinitis with chronic ethmoiditis and sphenoiditis. Exenteration of the ethmoid cells and the anterior walls of the sphenoids. Local anesthesia.

History.—Family history negative. No specific history obtained. Malaria fourteen years ago. Several attacks of otitis media. Dryness of the nose and crusting with loss of sense of smell for twenty yers.

Examination.—The middle turbinals are covered with dry crusts and also the postnasal space. The nasal cavities are wide. The turbinals are atrophied on both sides, including the middle and inferior. The mucous membrane is pale and dry. There are several erosions on the septum.

Result.—This case-was one representing the socalled terminal stage of ozena, and responded quickly to operative measures. For four years there has been no nasal symptoms.

Case 6.—C. N. C., Irish-American, born in Ireland, aged 53 years, married, news agent. First seen November 18, 1908.

Diagnosis.—Bilateral atrophic rhinitis with chronic ethmoiditis and sphenoiditis.

Operation.—Radical exenteration of ethmoid and sphenoid sinuses under ether.

History.—Family history negative. No specific or tubercular history obtained. Catarrh for ten years, with nasal obstruction and loss of smell. Colds often. Hacking cough. Postnasal discharge. Patient spends a long time each morning trying to get his nose free from crusts. Indigestion with chronic bronchitis and bronchorrhea for ten years. Aprosexia and neurasthenia marked. Odor pronounced.

Examination.—Face pale and pinched. Nose small and alæ flattened. Septum to the right. Inferior turbinates atrophied. Right middle turbinate large and covered with dry secretion. The left middle turbinate shows myxomatous degeneration, and there is a polyp between it and the outer wall. Erosion on the septum. Pharynx atrophic, glazed and dry.

Result.—Following a radical exenteration the crusts and odor disappeared, and for two years there have been no nasal symptoms.

Case 7.—M. F., Irish-American, born in Boston, aged 20 years, female, single, brushmaker.

Diagnosis.—Atrophic rhinitis with chronic bilateral sinusitis of ethmoid and sphenoid.

History.—Family history negative. No specific or tubercular history. Pneumonia ten years ago. For four years the patient has had headaches on rising, especially over the right eye. Colds often. She has been under treatment at various hospitals for four years. Sense of smell lost; occasional epistaxis. Odor marked. General condition fairly good.

Examination.—Alæ flattened, septum is deflected to the left. Both inferior turbinates are covered with crusts. Right middle turbinate very large. Much mucopus in the middle meatus. Mucous membrane, where it is not covered with crusts, is dry and the nose obstructed. Pharynx dry.

Operation.—Exenteration of ethmoid cells and anterior wall of the sphenoids under local anesthesia. Os planum on the right side is partially destroyed.

Result.—Patient has been seen at frequent intervals for a year. For the last two years she has been entirely well, with no nasal symptoms.

Case 8.—G. B., American, born in Vermont, aged 25 years, married, advertising manager for a machine company.

Diagnosis.—Atrophic rhinitis with bilateral sinusitis of ethmoid and sphenoid.

Operation.—Exenteration of ethmoid and sphenoid sinuses, under ether.

History.—Family history neagtive. No specific history obtained. Frequent colds. Loss of smell. Nose obstructed and dry for ten years. Scarlet fever as a child. Pneumonia ten years ago. General pressure symptoms over the entire head. Muscular weakness. Aprosexia. Odor marked.

Examination.—General condition poor. Face pale. Alæ flattened and dilated. Odor marked. Septum deviated to the right with a horizontal ridge and posterior spur. Both outer walls of the nares are covered with crusts, which bleed on removal. Middle turbinals swollen and ulcerated. Much mucopurulent discharge, especially in the posterior nares. Pharynx dry and glazed.

Result.—This case was much improved, but on account of the fact that he lived in an adjoining state, and could not be seen except at long intervals, he is far from being cured. Since treatment was stopped there has been no crusting or odor, but there is considerable purulent discharge, which the patient can remove by using a nasal wash.

CONCLUSIONS.

The illustrative cases of ozena here presented are only a few out of a large number seen by the writer during the last ten years. The study of these and similar cases has led to the belief that ozena is the sequel of a focal infection.

Clinically the course of events is as follows: A septal deviation causes a compensatory hypertrophy of the middle turbinal. This is followed by a chronic catarrhal ethmoiditis which interferes with drainage to such an extent that a subsequent active infection results in a sinusitis.

The fetor and crusting are probably due to the direct action of a specific pus producing organism on the tissues, without any preceding true atrophic process.

The difficulty in establishing free drainage, where there is an abnormal anatomic development of the bony cells, makes the cure of certain cases of ozena impossible with any intranasal procedure.

Whatever aid bacteriology may give us in curing this disease must follow the free drainage of all foci of infection.

BIBLIOGRAPHY.

Alexander, A.: Serodiagnostische Untersuchungen zur Frage der Beziehungen zwischen Ozaena und Syphilis. Ztschr. f. Laryngol. u. Rhinol., Wurzb., 1909, I, pp. 669-684.

Alexander, A.: Ueber das Wesen der Ozaena. Arch. f. Laryngol. u. Rhinol., Berlin, 1909, XXII, pp. 260-348.

Baumgarten, E.: Ueber the Frueformen der Ozaena. Arch. f. Laryngol. u. Rhinol., Berlin, 1909, XXII, pp. 492-497.

Betti, A.: Some Observations in Ozena. Revue hebd. de Laryngol., etc., November 12, 1894.

Compaired: Pure Atrophic Ozena of the Eustachian Tube and Middle Ear. Rev. Hebdom. de Laryngol. de Otol. et de Rhinol., October 15, 1910.

Dionisio: Radiation in the Treatment of Ozena. Recuel de memoires sur la Layngol. la Rhinol et l'otol., No. 2, 1904.

Dobell: The Formation of the Secretion in Ozena. Arch. f. Laryng., Vol. XV, No. 1, 1903.

Eckstein: Dauerheilung einer Ozaena durch Hartparaffininjektion. Berlin. klin. Wochenschrift, 1911, XVIII, pp. 401-403.

Eisenbohr: Die Wassermansche Reaktion bei Ozaena. Ztschr. f. Ohrenh., Wiesbaden, 1909, VII, pp. 401-410.

Frese: Ueber die Beziehungen der Syphilis zur Ozaena. Arch. f. Laryngol. u. Rhinol., Berlin, 1908, XX, pp. 459-474.

Freudenthal: Is Atrophic Rhinitis Always Autochthonous? Annals of Otol., Rhinol. and Laryngol., June, 1903.

Goswin-Ziehgraf: Ueber Behandlung mit Ozaenalyt. Klin. therap. Woch., Berlin, 1910, XVII, p. 1226.

Gruenwald and A. Waldrann: Studien ueber den bakteriellen Anteil an der Produktion des Ozaena Syndroms. Centralbl. f. Bakteriol. I. Abt. Jena, 1911, XX, Orig. pp. 337-350.

Gumaccia, Prof. Catane: The Paraffin Treatment of Atrophic Rhinitis. Rev. Hebd. de Laryngol., etc., October 15, 1904. Laryngoscope, p. 70.

Harris: Atrophic Rhinitis Depending Upon Ethmoiditis With External Operation on the Ethmoid. Laryngoscope, 1909, p. 548.

Hollinger: Ozena. No Terra Incognito. Laryngoscope, 1909.

Hopman: Sicheres und Unsicheres ueber Ozaena. Monatschr. f. Ohrenh., 1907, XXI; pp. 255-273.

Hutter, F.: Zur Paraffin Therapie der Ozaena. Arch. f. Laryngol. u. Rhinol., Berlin, 1910, II, XXIV, pp. 189-198.

Iglauer: The Treatment of Atrophic Rhinits by Oro-Naso- and Canula. Laryngoscope, 1906, Vol. XV, p. 833.

Kahn: Treatment of Ozena by the Submucous Injection of Paraffin Into the Nasal Chambers, With a Review of Other Methods. Laryngoscope, 1911, p. 737.

Kyle: The Pathology of Atrophic Rhinitis With Ozena. Laryngoscope, 1908, p. 423.

Large: Atrophic Rhinitis. Laryngoscope, 1909, p. 230.

Lautman: Ist die genuine Ozaena eine Infektionskrankheit? Ztschr. für Laryngologie, Rhinol., Würzburg, 1909, I, pp. 421-429.

Moeller: Atmokausis bei Ozaena. Verhandl. d. Ver. Deutscher Laryn., Wuertzburg, 1910, pp. 317-322.

Mueller: Ueber die Behandlung der Ozaena simplex mit Protargol. Zentralblatt f. d. ges. Therapie, Wien u. Berlin, 1908. XXVI, pp. 449-452.

Myles: The Treatment of Atrophic Rhinitis, Including Ozena. Laryngoscope, 1908, p. 425.

Rice: Atrophic Rhinitis and Ozena. Laryngoscope, 1908, p. 435.

Richards: The Treatment of Atrophic Rhinitis. Laryngoscope, 1908, p. 429.

Schmidt: Zur Therapie der Ozaena. Gurgeln mit geschlossenem Mund. Muenchen. med. Wochenschr., 1908, XV, p. 1239.

Schoenemann: Weitere Beitraege zur Pathogenese und Therapie der Ozaena. Verhandl. d. Ver. deut. Laryngologen, Wuerzburg, 1909, pp. 100-102.

Sobernheim: Wassermansche Komplementbindungsmethode und Ozaena. Arch. f. Laryngol. u. Rhinol., Berlin, 1909, XXII, pp. 1-9.

Sobernheim: Ozaena u. Syphilis. Arch. f. Laryngol. u. Rhinol., Berlin, 1909, XXII, pp. 430-435.

Steiner: Development of the Study of Ozena. Oroosi hetil. Budapest, 1908, II, pp. 622-624.

Steiner: Zur Weiterentwicklung der Lehre von der Ozaena. Arch. f. Laryngol. u. Rhinol., Berlin, 1908, XXI, pp. 282-290.

Stiel: Ueber die Behandlung der Ozaena mit Dionin. Therap. Monatschr., Berlin, 1907, XXI, pp. 633-635.

Theisen: A Consideration of the Herd Theory as an Etiologic Factor in Ozena, Laryngoscope, 1908, p. 417.

XVII.

INTRANASAL TREATMENT OF MECKEL'S GANGLION.*

By E. M. Holmes, M. D.,

Boston.

The treatment of the sphenopalatine as well as that of the Gasserian ganglion is almost always for the relief of severe neuralgic pain. It consists of removing the ganglion by an operation which is necessarily extensive and associated with considerable danger, of injecting some drug into the substance of the gland, or of making application through the nose to the mucous membrane overlying the area of the ganglion.

As it is of considerable importance in our present study, it seems advisable that we briefly review the history of the subject. In early medical literature we find much concerning the treatment of cranial and facial neuralgia. Practically every drug known at the time was tried and lauded by some writer. The hot iron, the wet and dry cup and cantharides were extensively used, but rarely with marked or permanent results. There have always been cases more or less self-limited which, with or without treatment, have ceased and never returned. It is not the purpose of this paper to consider the major surgical treatment, and practically nothing was accomplished except by major surgery until in the early seventies. In 1874 Bartholow reported considerable success by injecting chloroform into the nerve structures. In 1884 Billroth began injecting osmic acid. He made repeated injections through the skin without exposing the nerve. About the same time Neuber and Eulenberg adopted very similar methods. In 1889 Bennett began exposing the nerve trunks by blunt dissection and injecting directly into the nerve structures a ½ to 2 per cent aqueous solution of osmic acid. Wright, Murphy and others were heartily in favor of Bennett's methods, and advocated their trial before resorting to major surgery. Schlosser was the first to record the injection of alcohol into the nerve trunks and ganglia for the relief of pain. He began his investigations about the year 1900. Baudoin, Ostwalt, Levy and others, after much experience, were quite enthusiastic about the alcoholic injection into the ganglia. Dr. Sluder was the first to attack the sphenopalatine fossa through the nose. He tried various obtunding drugs in connection with the alcohol he injected, in order to avoid the excruciating pain which almost always followed the use of the alcohol alone. He finally adopted a 5 per cent carbolic alcohol, and this is the solution I have used in all of my cases. Dr. Sluder's work was very painstaking and thorough, and it was due to his paper upon this subject that over two years ago I began injecting the sphenopalatine fossa through the nose under the guidance of my nasopharyngoscope. By the aid of vision the method is so simple and exact, the discomfort to the patient is usually so slight and the results are often so gratifying, that I now report the findings of the work to this society.

---

*Read before the American Laryngological, Rhinological and Otological Society, Washington, May 10, 1913.

In this paper we have to consider two quite distinct classes of symptoms. First, the true tic douloureux, the distinguishing diagnostic symptom of which is a paroxysmal intense burning, cutting pain, lasting from a few moments to a minute or more. It may occur without known cause or it may be precipitated by various stimuli, as opening the mouth, swallowing, talking, sneezing and touching the face. A draught of air or a loud noise may bring on an attack. There may be from one to fifty attacks in a day. The attacks may follow day after day for years, or there may be periods of relief. There may be tenderness over the nerve foramina. In some cases there are vasomotor disturbances, flushing of the skin, hypersecretion of the nasal mucous membrane, salivation and lacrimation. The second class of cases is quite different in character. It includes the severe acute recurring hemicranias, the chronic almost constant hemicranias, the facial neuralgias and the deep boring suboccipital pains. A patient may be subject to one or all of the symptoms of the first class, to one or all of the symptoms of the second class, or symptoms of the two classes may be present at the same time. In this series some came with symptoms of the first class, some with symptoms of the second class, and some presented symptoms of both classes at different times or at the same time.

As regardless of symptoms there has been relief by the treatment of Meckel's ganglion, until we possess more definite knowledge, we can clinically, from the standpoint of treatment, consider them collectively. The important questions from the patient's standpoint are: What are the chances of relief? How severe are the treatments? How many treatments will be necessary to produce a cure or to receive the greatest possible relief, and how much danger, if any, accompanies the treatment? It will be seen by reviewing the history of the cases here reported that we can gain much knowledge, both as to the etiology and to the prognosis of treatment, by the application of a strong solution of cocain over the area of the sphenopalatine ganglion, for of the twelve cases relieved temporarily by the cocain, ten were later apparently cured by the alcoholic injection; and of the thirty-four cases not relieved by the cocain, only eight were cured and six were more or less relieved by the alcoholic injections. In the majority of the cases there has been, as a rule, slight if any pain during or after the injection, and therefore there has been but slight shock following the treatment. As yet we cannot answer the third question, for perhaps one injection may give permanent relief, or two or three injections may give no relief, while the next injection may give permanent relief, or may give relief for a short or a long period. A first injection may give relief for a few days, and a second may last for a few weeks, while a third may last for months and perhaps for years. It seems justifiable and proper to follow injections as frequently as symptoms recur until the condition is relieved. If there were no improvement after three injections, I would be inclined to consider the case hopeless, so far as alcoholic injections were concerned. We are, however, not in a position to positively state that a case is hopeless, even though three or four injections have been followed by no relief. Until I had treated my thirty-seventh case I had believed that there was probably no danger connected with the operation, but the alarming hemorrhage which began in this case on the fifth day following the injection, and coming, so far as we could learn, from or near the area of the injection, teaches us that we must not look upon the procedure too lightly. It is possible that this will not again recur in a thousand cases, but until I have found it absent in a very large number of cases, I shall always consider it, and probably may be inclined not to treat some who, though not suffering severely, ought to be given relief.

Before injecting Meckel's ganglion the nose and epipharynx should be carefully cleansed. The patient's head should be covered by a sterile rubber cap or towel. The face should be washed with alcohol. There should be in readiness a fresh sterile ½ per cent solution of cocain, a fresh sterile 40 per cent solution of cocain and a sterile Meckel's syringe filled with a 5 per cent carbolic alcohol solution. The nasopharyngoscope should be cleansed with alcohol. Although I have as yet not seen signs of sepsis in any of these cases, yet as it takes but a few minutes to carry out antiseptic measures, they, if not efficient, can do no harm and seem advisable.

After cocainizing the lower fossæ of the nose with the weak solution the nasopharyngoscope should be inserted into the side of the nose opposite to the ganglion to be injected, and should be pointed toward the side of that ganglion. The position of the ganglion, which varies somewhat, is approximately under the area just at the end of the middle turbinate, and at this place are almost always two or three small blood vessels seen emerging from the sphenopalatine fossa. A cotton tipped applicator soaked in the 40 per cent cocain solution is now passed through the same side of the nose as the ganglion we wish to treat, and when the tip is over the area to be attacked, the applicator should be held firmly in place. The nasopharyngoscope can now be removed and the applicator remain in place for five or six minutes. If the application has been made during an attack, we may learn much by the effects upon the symptoms. If the pain ceases or is much relieved, we are more likely dealing with a diseased or excitable ganglion, and in this case the chance of relief or cure is greater than when the cocain has no effect upon the symptoms. Some cases, however, which are not relieved by applications, are relieved or cured by the alcoholic injection. We are, therefore, I believe, justified in injecting all cases which are of the type suggesting ganglion disturbance.

After sufficient time has elapsed to thoroughly anesthetize the area through which the needle is to be inserted, the nasopharyngoscope should be again passed into the opposite side and the sphenopalatine area brought into view. My Meckel's ganglion or some other suitable syringe should now be passed through the side of the nose corresponding to the ganglion to be treated. The needle must be concealed within the canula. The canula tip must be directed by the aid of the nasopharyngoscope until it is in contact with the area into which the needle is to be thrust. It should be pointed outward and somewhat upward. The canula must now be firmly held and the needle pushed through it to the desired depth. When we feel that the needle point is in in the desired position, the alcoholic solution can be injected. I begin with two drops at the first injection and increase the amount a drop at following injections. Six drops is the most I have used at any time. Following the injection in some cases there is for a few seconds to a minute or two pain, which is frequently followed by a numbness in the face and in the palate. Some cases have no pain following the injection, some have immediately a numb tingling sensation in the face, and others show no effects at the time, but show results after a few days. Among the failures there are likely many in which we are not successful in striking the ganglion.

All of the cases in this report are of a chronic type. Many of them give histories of a number of years' duration. Some of them are of intermittent character and have had periods of comparative freedom from pain. It will of course be necessary in the intermittent cases that considerable time elapse before we can consider them as cured. I am including all cases in this study, for a number of them in this latter group were extremely intense during the attacks, and in some the attack was almost immediately relieved by the treatment.

In all there are forty-six cases, and the time limit will not allow us to report them individually or at all fully. It has therefore seemed advisable to give a general summary of the treatment and results, and to append sufficient of the case histories to illustrate some of the more important conditions. There were thirty-one females and fifteen males. The oldest patient was a woman 74 years of age. The youngest was a boy 17 years of age. The average age was a little over 41 years. The first case treated was about twenty-six months ago, and the last case was treated over two months ago. The number of injections in each case varies from one to nine, and the average for the series is just over three. Of the cases which were relieved by applying cocain 40 per cent solution over the area corresponding to the location of Meckel's ganglion (twelve in number), ten were relieved by the alcoholic injection and two were not relieved or in the least affected. Thirty-four cases were not relieved to any degree by the application of cocain to the area of the ganglion. Eight of these, however, were cured by the injection and six were more or less relieved. There were twenty-two cases which were not relieved to any marked degree by the injection. It is hardly fair to the treatment to give these failures without a few words of explanation, for some of them do not belong to this classification, but were treated experimentally, hoping to give relief to severe suffering. I do not believe now that injection of the ganglia was contraindicated in these severe cases, for the chance of giving relief to a distressing condition far outweighed the slight chance of untoward results. Among the relieved were eight cases of true tic. Five cases presented mixed symptoms, and eleven cases suffered with neuralgic pain not of the sharp, darting type. Of those unrelieved, there were three chronic suppurative sphenoid cases, four postoperative frontal sinus cases, two syphilitic necrotic cases, one glioma of the hypoglossal nerve, one calcareous degeneration of a second superior molar, and one case suffering severely from cicatricial contractions following an enucleation of a tonsil some three years previously. This last patient had been operated upon by a competent laryngologist, but evidently sepsis followed, resulting in very marked deformity. By plastic operations I was able to relieve the marked deformity, but there remained a constant painful dragging sensation. The first injection gave temporary relief, but following injections gave no results.

Although it is impossible to give a full report of all the cases, it seems advisable to append the histories of a few, as it will give a better understanding of the subject that I can by any general remarks.

No. 6 came to me first in June, 1910. She was 73 years of age and had suffered for a number of years with neuralgia. For two years she had been an almost constant sufferer with frequent intense jabbing pain, worse in the left side of the inferior jaw. She had been unable to eat without precipitating an attack. Her sleep had been greatly disturbed. She was emaciated and had a very haggard appearance. She had taken various drugs without relief. Hot applications to the affected area and absolute rest seemed the only measures which gave any relief. I at first tried injections into the mental foramen and into the nerve trunks, but without any apparent result. She suffered severely until February, 1911, when I injected carbolic alcohol into the sphenopalatine area. She complained of a steady pain for several hours and then was absolutely free from any pain for three weeks, when the darting pains began. They were nowhere near as severe, but she was anxious for another injection. Another alcohol injection was made at this time and there was no pain until the last of September, six months after the last injection. Another injection was made and there has been no recurrence of pain. The patient has gained in every way and she is now as well as any one of her age.

No. 37, a woman 33 years of age, began having attacks of pain in the head in September, 1911. These attacks lasted several weeks and then subsided. In February, 1912, she began having attacks of severe neuralgia in the right side of the face. The pain usually started in the right cheek and darted toward the right ear and occiput. The pain was at times steady and at times was accompanied with severe cutting, burning thrusts. Before having any trouble with pain she had an attack of catarrh of the right side of the nose, accompanied with much thick yellow discharge. Examination showed a deflection of the nasal septum to the right with a sharp spur about two-thirds back, pressing into the middle turbinate. There was no sign of pus or hypersecretion of any description. As relief from the nasal deformity was demanded, I performed a submucous operation. For a while after this operation the pain seemed less and the improvement, though slow, was steady. In October the old symptoms became worse. The attacks became frequent, and on the 19th an injection of carbolic alcohol was made. On December 7th patient said pain was much less but was still annoying. Another injection was made, nothing unusual happening until five days later, when there was a protracted and rather profuse hemorrhage from the right side of the nose. Dr. French of Lynn, who first sent the patient to me, was called and found a severe hemorrhage and the general signs which accompany considerable loss of blood. He was able to stop the severe flow by plugging the nose posteriorly and anteriorly. Even with these very tight plugs there remained a slight oozing. I saw the patient in consultation on the 14th. As there were signs of sepsis we tried to change the plugs. Great care was used in removing them, but the moment the anterior ones were removed the hemorrhage began. The blood was little different from colored water. Fresh aseptic plugs were immediately inserted. The patient was placed in a semiprone position and was given all the gelatin she would take. She remained in practical collapse most of the time until the 17th, when I again saw her. The plugs were of course getting very foul and had to be removed. The hemorrhage was the same as before. Only a moment elapsed before new plugs were in place. We had from the first considered transfusion as well as intravenous rabbit's serum. It was decided advisable to give rabbit's serum, and it was injected by the family physician. Two days later there was no oozing and the plugs were removed without any hemorrhage. We don't know what happened in this case, but can reasonably believe that the alcoholic injection was the exciting factor. There may have followed some necrosis which destroyed an arterial wall. We could prove nothing, for when we saw the case the hemorrhage was from a number of places, and when Dr. French first saw the case there was so much blood that he could not tell positively its origin. I am reporting this case as it is an important factor when considering this subject. A similar case may never again recur, but until a very large number have been followed we must not ignore this warning.

No. 24 is reported individually, as it illustrates a case showing all the symptoms of sphenopalatine neuralgia, but is really of another type. This patient, a woman 50 years of age, had been a sufferer for over twelve years. For a number of years there had not been a day when there had not been severe cutting pains starting on the left side of the nose and the left temple and radiating into the ear, the suboccipital region and into the neck. There had been several intranasal operations and there had been two major operations for the removal of Gasserian ganglion. The last operation was followed by facial paralysis and marked deformity, but with no relief from the excruciating pain. There had been three injections of alcohol into the sphenopalatine area from the outside, but with slight, if any, relief. In August last I injected the sphenopalatine area intranasally. This gave some relief for a few days. In a month there was as much suffering as ever, and she informed me that there had always been a very sensitive point on the left side of the throat down by the roots of the tongue. Inspection showed nothing, but examination with the finger showed a small hard submucous growth which was extremely sensitive. Slight pressure was always followed by a paroxysm of severe pain. I endeavored to anesthetize with cocain and remove the tumor, but found it impossible. The patient was then admitted to the St. Elizabeth's hospital, and under ether I removed the growth, about the size of a pea. The pathologic report was that the growth was a glioma. Following this operation the attacks were less and less, and there has been absolute freedom during the past three months. This case was a failure so far as the injection of alcohol was concerned, for although the symptoms would place it as a sphenopalatine complication, the etiology was very different.

No. 31, a man aged 48 years, had for a year and a half been a great sufferer with tic douloureux. About five or six years before the attacks there had been a profuse discharge from the right side of the nose, but no marked nasal symptoms since. For over a year he had been treated by various men at various hospitals. He had also been treated as a private patient by several. He had been unable to receive any relief. The attacks were occurring every few minutes, so that he had been unable to perform any work for several weeks. Inspection by the nasopharyngoscope showed the area over the right sphenopalatine fossa somewhat more prominent than usual. It was more prominent than the left side. Although this was noted, we were not at this time impressed by the condition. After injecting the fossa and removing the needle there was a discharge of foul smelling pus. Exploration showed a general necrosis of the surrounding bone. A Wassermann of the blood was strongly positive. He was given salvarsan, and was somewhat relieved for three months, when the pain returned and was intense as before. I again injected the sphenopalatine area, and the attacks disappeared for three weeks, when the injection was repeated. After another four weeks there had to be another injection. Since then there has been no return of trouble.

Although we have as yet not had sufficient experience to treat this subject as positively as it should be, both Dr. Sluder's experience and that gained from the observation of this series of cases proves that in a certain number of these distressing conditions we can temporarily give relief by applying strong cocain over the area of the sphenopalatine ganglion. In the cases thus relieved we may also expect marked results from the carbolic alcohol injections of the ganglion. Those cases not at all relieved by the cocain applications are not hopeless, for a few of them have been permanently relieved by the alcoholic injection. We have demonstrated that the procedure is a comparatively precise and as a rule painless one, and can be repeated a number of times if necessary. Our one case shows that alarming symptoms can follow this treatment, and therefore we cannot as yet use it in slight cases.

XVIII.

BRONCHOSCOPIC REMOVAL OF A COLLAR BUTTON AFTER TWENTY-SIX YEARS' SOJOURN IN THE LUNG.

By Chevalier Jackson, M. D.,

Pittsburg.

Bronchoscopic removal of foreign bodies are now so common that they scarcely merit publication, but the case herein reported presents unusual features.

Mrs. C. K., aged 48 years, admitted to the Presbyterian Hospital, with a history of having "swallowed" a pearl collar button twenty-six years previously. There was some cough and bloody expectoration at the time of the accident and for about a year subsequently. This was before the discoveries of bronchoscopy and roentgenoscopy. No further pulmonary symptoms were noted for twenty-four years. During the twenty-sixth year there was an atatck of "pneumonia," in treating which the attending physician (a very competent man) ridiculed the patient's idea that the button could still be in the lungs. In the early part of the twenty-sixth year, under care of a third physician, a second attack of pneumonia occurred, followed by a pain in the left side, bloody, foul expectoration, fever and emaciation. Again the patient's story of the collar button was ridiculed. Extremely feeble and emaciated, she fell into the hands of Dr. S. B. Pierce of Johnetta, Pa., who at once, on the physical signs, made a diagnosis of lung abscess and pyopneumothorax. Suspecting foreign body origin, he referred the case to me. On admission to the Presbyterian Hospital the woman's temperature was 102,° pulse 140, respirations 40. Sputum was profuse, thick, foul and of dark gray color. A radiograph (Fig. 1) by Drs. Johnston and Grier showed a dense shadow over the left lung, which they believed to be pus. Dr. John

W. Boyce corroborated Dr. Pierce's findings, and urged immediate drainage of the pleura by rib resection and a wide opening. This was done by Dr. J. Hartley Anderson. Johnston and Grier then made another anteroposterior radiograph (Fig. 2), which showed that the pus was well drained. In further search they made a diagonal radiograph which showed a collar button between the heart and spine, in direct line with the stem bronchus of the left side. With the assistance of Drs. Patterson, McCready and McKee, without anesthesia, general or local, I passed a bronchoscope and found the inferior lobe bronchus occluded, just below the orifice of the upper lobe bronchus, by a cicatricial mass containing three apertures through which reddish granulations, that bled when wiped, were protruding. Clearly, dilatation, as practiced in previous cases, was useless, and with the accurate localization and advice of Dr. Johnston as a guide, I excised tissue until a rather large cavity full of granulations was reached. I excised the granulations and wiped away blood, foul pus and secretions until the collar button (Fig. 3) came into view, bedded in granulation tissue, from which it was readily removed. The foul odor disappeared in about four weeks, cough and expectoration lessened, and both have now disappeared. The lung has filled with air and the woman is well.

The points of special interest are:

1. The extremely long sojourn of the foreign body in the lung; the longest, I believe, yet recorded.
2. The freedom from symptoms after the first year, for so long a period, twenty-four years.
3. The bursting of a foreign body abscess into the pleura, while doubtless not of the greatest rarity of occurrence, has been recorded in only a few instances.
4. The foreign body did not follow the discharging abscess into the pleural cavity.
5. The necessity of the most expert ray work is imperative. It was only the lateral radiograph that could show this foreign body, and ordinary work would have been negative.
6. The necessity in such cases of draining pus collections in order to get a radiograph of a foreign body, which would not show through the purulent shadow.

7. The feasibility of endoscopically removing a tissue barrier in order to reach an abscess cavity in the lung, when guided by both an accurate radiographic localization and ocular evidence that the tissue to be removed is pathologic.

8. The advantage of working without an anesthetic. This patient was in extremis and an anesthetic was not to be thought of. Moreover, it was no more painful than the filling of a sensitive tooth cavity, for which no one requires an anesthetic. Furthermore, the air passages were full of pus mixed with blood from the granulations. The coughing of the unanesthetized patient greatly assisted in removing this in the way I have often elsewhere explained.

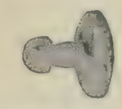


FIGURE IV.

Glass collar button removed from the lung by bronchoscopy bloodlessly through the mouth without any anesthesia, general or local. The collar button had been in the lung for twenty-six years.

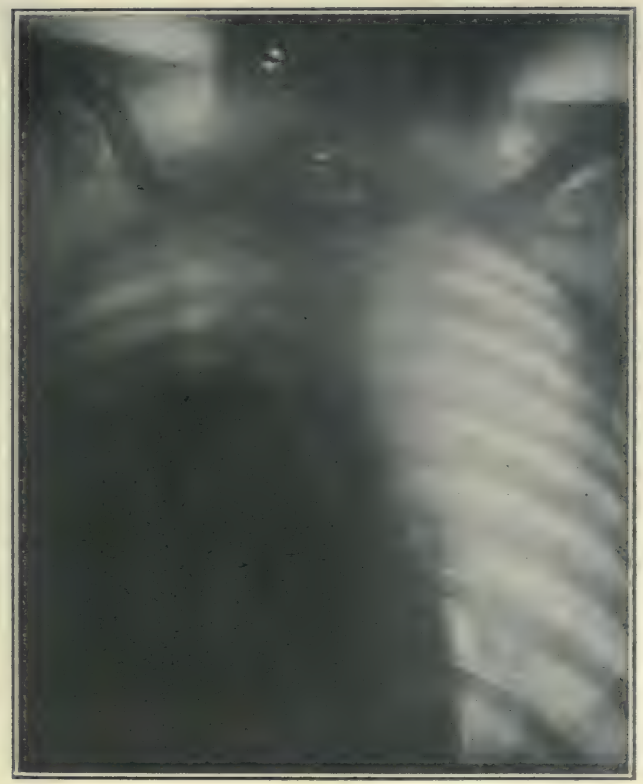


FIGURE I.

Radiograph showing left pyopneumothorax which was due to the bursting into the pleura of a foreign body abscess of the lung in a woman of forty-eight years. Collar button was in the lung, but did not show through pus shadow.

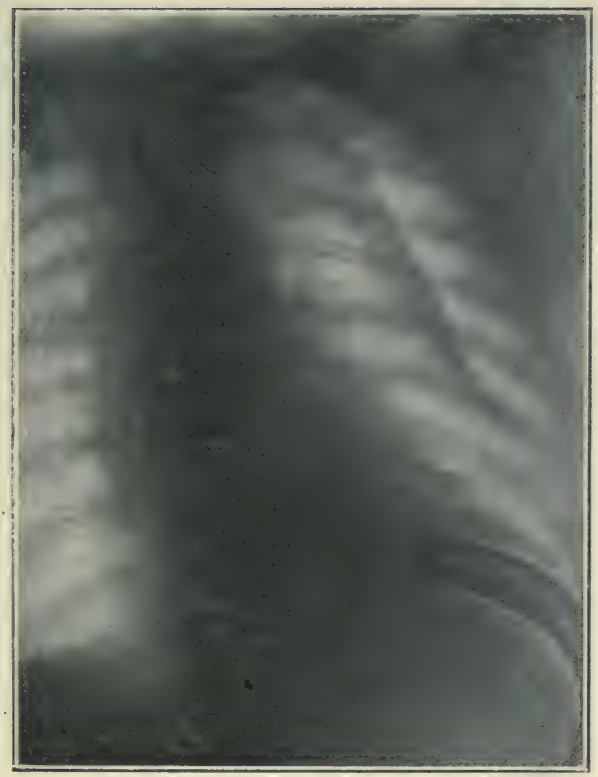


FIGURE II.

After external drainage of the abscess by Dr. J. Hartley Anderson, foreign body (collar button) present did not show. The dark line from the first rib downward and outward toward the drainage tube is the thickened visceral pleura seen on edge. The lung is collapsed as far as the pleural adhesions will permit.

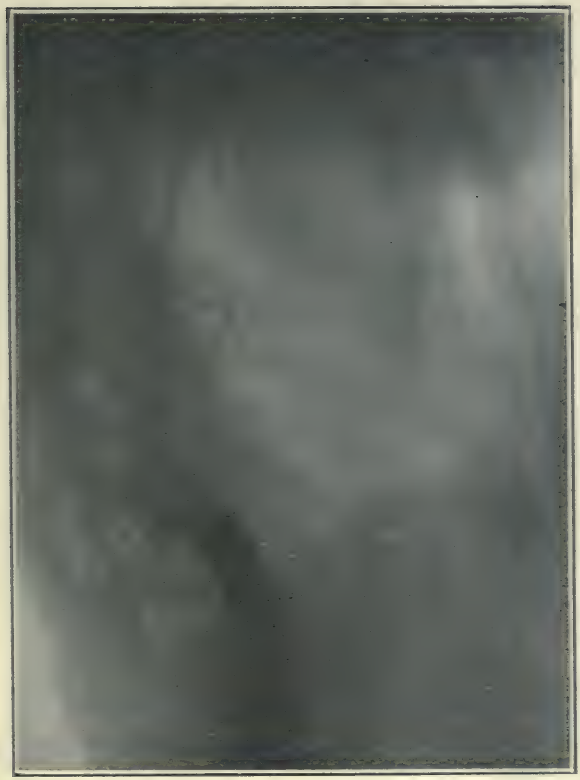


FIGURE III.

Quartering lateral radiograph by Dr. George C. Johnston, showing collar button in the lung between the heart and the spine.

XIX.

THE COMBINED LABORATORY AND X-RAY INDICATIONS FOR THE MASTOID OPERATION.*

By George Sloan Dixon, M. D.,

New York.

For many years it has been the routine practice in the Eno Laboratory of the New York Eye and Ear Infirmary to examine smears from the aural canal in every case of suppurative middle ear disease, acute or chronic, admitted to the wards. For a short time cultivations were made, but these were soon abandoned, as the germs present in the canal are always mixed, and much time was wasted in separating them. As the mastoid pus in cases coming to operation was also examined, comparison showed that the predominating germ in the canal was found in the mastoid pus with sufficient frequency to make it a good rule to regard the predominating germ in the discharge from the middle ear responsible for the mastoid involvement, and we have found no good reason to change this view. For example, it is the rule to find the predominating germ shown in the smear from the canal pus, in the pus from the middle ear, in practically pure culture in the mastoid pus, in the pus of complicating brain abscess, in the spinal fluid in cases complicated by meningitis, and in the general circulation in cases complicated by sinus thrombosis.

During the past eleven years we have made 5496 examinations of aural pus. Of these 26.6 per cent have been mixed infection; that is to say, a large number of mixed germs were present, and it was not advisable to charge the infection to any particular germ. The majority of these were chronic cases, and where a radical mastoid operation was done the mastoid smear usually showed mixed germs. Streptococcus occurred as the predominating germ in 24.8 per cent; pneumococcus, 12.2 per cent; staphylococcus (aureus and albus), 7.4 per cent; streptococcus mucosus capsulatus, 4.8 per cent; spirillum of Vincent, 1.8 per cent. The remaining 22.4 per cent is represented by miscellaneous, negative, etc., among which are placed the bacillus pyocyaneus, diphtheria, tubercle, coli communis, Friedländer, etc.

---

*Read before the Medical Association of Greater New York, February 17, 1913.

The discrepancy as to the frequency of appearance of these various forms of infection, as compared with the reports of other workers, is only apparent, and is largely accounted for by the fact that no distinction has been made between acute and chronic suppurative otitis and their complications.

In the matter of the relative virulence of these various microorganisms, our experience leads us to regard the streptococcus mucosus capsulatus as standing first, streptococcus pyogenes next, pneumococcus third and staphylococcus fourth, in acute suppurative processes about the middle ear.

At this point I wish to reiterate a statement made a number of times, to which exception has been taken, and will probably be again, namely, that among the various microorganisms found in the middle ear the streptococcus mucosus capsulatus in a manner stands alone. It is the most insidiously destructive the otologist is called upon to combat. This time it is my desire to make the statement as plain and as emphatic as possible.

A case of acute purulent middle ear disease in which the infective germ is the streptococcus mucosus capsulatus will tend to recovery equally with other forms of serious infection, provided it is seen early and proper drainage established. It is not these cases particularly to which reference is made, though such should immediately put the surgeon on his guard. It is those cases which are neglected; or cases in which all the acute symptoms have subsided but the otorrhea persists and the streptococcus mucosus is present, to which attention is directed.

Such a case may lull both the patient and surgeon into a sense of surety against further unfavorable progress. The pulse and temperature may become normal, all pain and tenderness may disappear. The blood count may return to normal. The hearing may improve, but the otorrhea does not cease, as a general rule. Cases of this nature demand at least an exploratory mastoid operation. As a rule, every day over two weeks' duration is an additional and increasing menace to the life of the patient.

CASES OF STREPTOCOCCUS MUCOSUS CAPSULATUS INFECTION.

Case 1.—A male, 22 years of age, in the service of Dr. Adams, was admitted to the infirmary August 10, 1910. He gave a history of having had pain in and discharge from both ears for about twelve days. He was under observation and ran a temperature ranging from 98° to 100° until September 14th (twenty-nine days), when he complained of headache, and next day he had a chill and the temperature rose to 103.2.° During the time he was under observation two blood counts and a blood cultivation were negative. Mastoid operation was done September 15th. The bone was found extensively necrosed, there was marked zygomatic development and all cells were filled with pus. The dura was exposed in the middle fossa. Meningitis occurred, and the patient died six days later.

Case 2.—Male, 39 years of age, in the service of Dr. Whiting, was admitted to the infirmary September 8, 1909. He gave the history of having "snuffed up salt water" two months before, which caused great pain and apparently rupture of the membrana tympani. This cleared up in about two weeks. His present attack began eleven days ago, with great pain but no discharge. Now has frontal headache. A Schwartze operation on the day of admission. An escape of pus followed the skin incision. A large perforation was found at the root of the zygoma, which was followed into the zygomatic cells. Bone was disintegrated and removed with a curette. There was also found a perforation through the dural plate into the cranial fossa. The patient died of meningitis five days later.

Case 3.—Male, 39 years of age, was admitted to the service of Dr. Kenefick, February 26, 1912, with a history of having had coryza for twelve days, with a violent headache on the left side. The pain became localized in front of ear, and he had a seropurulent otorrhea for twenty-four hours; had pain in the mastoid which gradually subsided, and all acute pain ceased. Examination on admission showed the right ear normal. The left ear showed subsiding O. M. P. A. There was no drooping of the canal wall, but there was tip tenderness in mastoid. Had aphasia and other symptoms of temporosphenoidal abscess. The patient was operated, but he died two days later.

Case 4.—Male, 28 years of age, was admitted to the service of Dr. Bacon, November 4, 1910, with a history of his present illness having lasted three weeks. He had had discharge from right ear, headache and mastoid pain. Had steadily refused to enter infirmary.

When he came in, pus was found in the canal, there was an anterior perforation in the membrana tympani which was red and bulging, and there was sagging of the superior posterior wall of the canal. He had marked mastoid tenderness, temperature 105.6,° pulse 160, respirations 24.

Mastoidectomy was done same day. A large area of sinus and dura were exposed, which were covered with granulations and pus under pressure. The patient died of meningitis next day.

Case 5.—Female, 40 years of age, was admitted to the service of Dr. Kenefick, with a history of having had slight pain in the right ear followed by otorrhea two months previously. This continued for three weeks, and then apparently ceased. A week ago was seized with severe pain, had chilly sensations and temperature, but no discharge. When she came in the canal was red and dry, bulging of membrana tympani. The posterior superior canal wall was sagging. Myringotomy, followed by a profuse discharge of blood and pus. Next day, July 23, 1909, Dr. Yates did a Schwartze operation on the right side. The mastoid was found sclerotic—very few cells. The bone was soft about the sinus and was removed. A small area of dura in the middle fossa was exposed. Progress was uneventful and she was discharged August 2d. Six days later she was readmitted at night, with the history of having had severe headache, nausea and vomiting, also vertigo, and became comatose. When she came in her temperature was 104,° neck stiff, eye grounds normal. Spinal fluid contained streptococcus capsulatus. An exploratory operation was done the next morning and a thrombus removed from the right lateral sinus. The patient died of meningitis next day, two months and nineteen days from the beginning of her ear trouble.

Case 6.—Male, 39 years of age, was admitted to the service of Dr. Dench, June 28, 1909, with a temperature of 104.° The history given was that five weeks previously had pain in the left ear; a week later it began to discharge. This continued for two weeks, then ceased entirely for a week. Later had severe pain for a few days until the drainage was reestablished. A Schwartze operation was performed the day of admission. The cortex was hard, there were few cells, and the bone was soft in spots. The cells present contained granulations. The sinus was exposed for about an inch. The wall was white, but there was free flow of blood when the sinus was opened. The temperature dropped immediately, and he was discharged in good condition, twelve days later, and ordered to the clinic for further treatment. At the August 16th dressing, which was six days after he had left the ward, there was a very slight hemorrhage, also one on the 20th a little greater, and on the 23d there was a gush of blood from the sinus. The patient was readmitted and sent to the operating room. After opening the old wound and exposing the sinus widely, pressure was applied and the patient was put to bed. The next day neuroretinitis was found on both sides, but he did very well until September 4th, twelve days after his second admission, when he began to run an irregular temperature. The blood counts at this time were practically negative, but four days later (September 8th) a positive blood culture was obtained—streptococcus capsulatus. The internal jugular vein was resected the same day, and a cultivation of the contents of the vein yielded streptococcus capsulatus also.

Further progress was rather uneventful until November 23d, an interval of eleven weeks after his internal jugular resection, when he was discharged, his pulse and temperature having been practically normal for over three weeks.

On December 10th, seventeen days after his discharge from the infirmary for the second time, he complained of not feeling well while in the clinic, and was directed to return that afternoon. The night of the 10th (that is, next day) word was telephoned to the infirmary that he had not been able to return, as requested. Two days after this he was brought to the infirmary at 8:30 p. m. with meningitis. He was taken to the operating room and an attempt made to drain, but he died in four hours. The total duration of this case was from about the 24th of May to the 12th of the following December.

These few cases have been picked at random. They illustrate the point, and it is therefore unnecessary to tax your time and patience further. But we have records of plenty more of the same kind.

True, other forms of infection may be equally dangerous, as, for example, streptococcus pyogenes and pneumococcus, but in these there is little likelihood of such a complete cessation of symptoms, and the danger signals are pretty constant.

In cholesteatoma the finding of cholesterin crystals in specimens from the canal or attic may aid in the diagnosis, though it is generally possible to make a diagnosis without the aid of the laboratory; however, our services are occasionally useful. In our experience the microorganisms present in this form are of little importance, as a rule.

BLOOD EXAMINATION.

The blood count in simple acute mastoiditis will range from normal to 18,000, with an average of about 11,000 to 12,000 leucocytes. The polynuclear count may be as high as 80 per cent, but the average is about 70 per cent. A leucocytosis of over 20,000 generally indicates the presence of some complication, meningitis, for instance, while 25,000 or over is more likely to indicate pneumonia. A leucopenia may be among the first indications of typhoid fever as a complication. We have, however, seen a leucocyte count as low as 4,200 with a polynuclear count of only 48 per cent in meningitis, but this is unusual.

Eosinophiles and mast cells are generally absent in cases of acute mastoiditis.

The blood count may be an excellent indication of the resistance of a patient or the onset of complications, but gives little information of the condition of the mastoid itself.

The time at our disposal will not permit of considering the complications of mastoiditis, but we may say a word about blood cultivations. In blood cultivations we have an excellent means of determining whether the patient is suffering from bacteremia or not, and a positive cultivation in the presence of middle ear suppuration may be taken as good evidence of thrombosis of the lateral sinus, provided all complications of a suppurative nature below the neck can be eliminated. It has been stated that meningitis will occasionally give a positive blood culture. This may be true, but no case of meningitis coming under our observation has yielded a positive blood culture where implication of the lateral sinus could be excluded.

RADIOGRAPHY.

If any advantage is to be derived from mastoid radiography, we must of course have some knowledge of the variations in the normal mastoid process. We all know that a mastoid process may contain pneumatic cells of the medium, small or large type; that the small cells are in the upper, and the large cells in the lower mastoid, as a rule; that we may find coarse or fine diploetic bone, or solid bone throughout, only the aditus and antrum being present to be regarded as pneumatic spaces, and sometimes they are exceedingly small. Finally, the cortex may be very thick or very thin, and combinations of any two or all of the varieties may occur on one or both sides, except that the cortex is more likely to be the same on both sides. The size and shape of the mastoid are likely to be symmetrical.

Mr. Cheatle exhibited an interesting series of one hundred and twenty paired temporal bones at the Ninth International Otological Congress, held at Boston last August, and his demonstration was very valuable, particularly from the radiologist's point of view.

For instance, the right lateral sinus is usually larger and further forward than the left, and further forward in diploetic than in cellular bones; but in his series 21 per cent showed the groove larger on the left, and 64 per cent of these were males. In other respects there were exceptions, but not a sufficient number to change the rule. In any event, for our purpose it is not so much the size of the sinus as the depth of the groove and the prominence of its anterior margin.

More important is the fact that 16.6 per cent of these bones showed the diploetic infantile type on one side only. The total cellular asymmetry was 31.6 per cent, the other 15 per cent being of asymmetrical arrangement and combination of cells and diploe in the upper and lower mastoid. Thus, speaking broadly, the mastoid process was symmetrical in 68 per cent of his specimens.

This bears in an important manner on radiography, and must be considered when reading the plates.

We are thus met at the outset with difficulties, but they are more apparent than real, and in practice we can often distinguish the fine lines marking a diploetic process as distinguished from an empyema which may have obliterated the cell outlines; but of course this is not always possible, for we are dealing with shadows, and not with what we ordinarily understand as pictures. We have noted a number of cases where the mastoid on one side was diploetic and of the pneumatic type on the other, the pneumatic side being the side involved, while there was no history of disease on the diploetic side.

In view of this, it may very properly be asked what we may reasonably expect in the way of information to be obtained from an X-ray examination of the mastoid.

First: It will show in the vast majority of cases the location of the lateral sinus, judged by the shadow of the anterior margin of the groove in its relation to the meatus, and its angle to Reid's base line, which is most important. If this shadow of the anterior margin of the groove is found to be at right angle to Reid's base line, the sinus has cut the petrous pyramid and must necessarily be very close to or immediately under the posterior canal wall, and as this angle decreases backwards the sinus is further and further from the canal. If it cuts Reid's base line at about 50 to 55 degrees it may be regarded as about in its normal position. The possibility of obtaining this information is alone sufficient to warrant an X-ray examination.

X-RAY CASES.

Case 924.—F. 42. Dr. E. B. Dench. Furunculosis with suspected mastoiditis on the left side. Skiagraph showed the mastoids diploetic on both sides with sinus far forward.

Three days later a simple mastoid operation was done. The sinus was found to be superficial and very close to the posterior canal wall. The mastoid antrum was explored through the canal, and no cells were found except at the tip.

Case 937.—F. 16. Dr. E. B. Dench. Left O. M. P. C. with furunculosis. Skiagraph showed a few small cells on the right side, with sinus in about the usual position. No cells were apparent on the left side, and the sinus was far forward. Left radical mastoid operation was done. The bone was found to be dense, and the sinus far forward.

Case 1206.—M. 23. Dr. E. B. Dench. Right furunculosis with suspected mastoiditis. Skiagraph showed the left mastoid having the appearance of being between a fine pneumatic and diploetic type. The cells which could be seen were well defined above and behind the canal, and could be traced to the tip. The right mastoid showed a few ill defined cells above and behind the canal, but could not be traced below the level of the canal. The lateral sinus on this side was observed to be about 15 degrees straighter than the left—that is, well forward. The whole mastoid was cloudy. Simple mastoid operation was performed the next day on the right side. The mastoid was reported as diploetic, with the sinus against the posterior wall of the canal. No free pus was found, but the antrum was filled with granulations.

Case 1503.—M. 34. Dr. Kenefick. Left O. M. P. A. Mastoiditis. Skiagraph showed both mastoid processes of large cell pneumatic type, the right clear, but the left cloudy throughout. Sinus fairly well forward and deep, being covered with cells. Four days later a simple mastoidectomy was done on the left side. The cells were found filled with pus and granulations, the sinus far forward and deep.

Case 1505.—M. 29. Dr. J. L. Adams. Left O. M. P. A. Mastoiditis. Skiagraph showed both mastoids of markedly diploetic type—no cells to be seen. The sinuses were far forward. Simple mastoidectomy the same day. The bone was found to be sclerotic. Sinus was extremely far forward, and just below antrum curved forward under posterior canal wall—almost in contact. Tip diploetic. Pus was found in the antrum.

Case 1563.—F. 10. Dr. W. S. Bryant. Right O. M. P. C.

Skiagraph showed the left mastoid to be of pneumatic type, moderate sized cell. Clear. Right showed only a few indistinct cells over sinus, which was far forward. Radical mastoid operation next day. No cells were found, the bone being diploetic. The bone was soft and full of granulations. The sinus was far forward.

Second: We may expect to learn the type of bone, the location of the large cells, whether the sinus is covered with cells or not, and the extent to which they have developed into the zygomatic region. If cells do not show on the diseased side and do show on the normal side, the chances are about three to one that the pathologic bone is of the same type. If no detail is found in the plate of the affected side, then the chances are about five to one that the mastoid is diseased; but the clinical symptoms must back up the plate.

Case 977.—M. Dr. E. B. Dench. Suspected mastoiditis. This patient had been walking about for some weeks. He was a man having important business connections, and it was quite necessary that he remain in harness unless an operation was absolutely necessary at that time. He was apparently convalescing from an O. M. P. A. right side, and was comfortable except for the discharge. Skiagraph showed left mastoid of large cell pneumatic type, and clear. The right was disorganized and appeared like a cavity on the plate and was so reported. Soon after this a mastoidectomy proved the findings as above.

Case 1024.—M. Dr. E. B. Dench. Right O. M. P. A. Mastoiditis. Had intranasal operation three months ago, and has had more or less ear trouble ever since. Skiagraph showed pneumatic mastoids on both sides. The right was cloudy. Sinus was not shown. Simple mastoid operation two days later. Free pus in tip cells.

Case 1090.—M. 22. Dr. R. Lewis. O. M. P. A. Mastoiditis right. Skiagraph showed both sides of the large cell pneumatic type. The right was very cloudy. Sinus in about the usual position. Simple mastoid operation was performed three days later. A cellular bone was found, the tip cells filled with pus.

Case 1349.—M. Dr. E. B. Dench. Left O. M. P. A. Deaf on left side for several years. Had a great deal of pain three weeks ago. Paracentesis gave relief. No history of mastoid tenderness. The infection was mixed, streptococcus capsulatus being present. Skiagraphs showed right mastoid with small cells about petrous base and antrum. A large cell was shown about the middle of mastoid, divided by anterior margin of sinus groove. Left mastoid was cloudy throughout—dark where the cells of lower mastoid should be, and septa indistinct. Left simple mastoidectomy. Mastoid extremely pneumatic, with numerous large cells at tip full of granulations and pus.

Case 1351.—F. 20. Dr. Gorham Bacon. Left mastoiditis. Skiagraph showed mastoid of moderate sized pneumatic cells which extend high above canal. Left same type. The higher cells on this side very cloudy. The cloudiness shades off to tip. Simple mastoidectomy the same day. Free pus with granulations found throughout the mastoid, especially at antrum and tip.

Third: It is possible to diagnose perisinus abscess. I have been fortunate enough to diagnose one, hint at others, but have missed several. More experienec and improved technic should increase the number discovered. Epidural abscess should be diagnosed, if it is not in line with the sinus, in a small proportion of the cases at least.

Case 942.—M. Dr. E. B. Dench. Suspected mastoiditis. Skiagraph showed left mastoid process involved. The plates also showed the shadow of an epidural abscess distinctly, but this was not read before operation. Two days later simple mastoid operation was done. Thick and hard cortex was found. Free pus was found in antrum and tip. Epidural abscess over cerebellum.

Case 987.—F. Dr. E. B. Dench. Suspected empyema left mastoid. Skiagraph showed small pneumatic type of bone on both sides. A small spot in front and one behind the left sinus were thought to be cells. Operation next day demonstrated the two small spots to be perisinus abscesses.

Case 1235.—M. Dr. Gorham Bacon. Right O. M. P. A., suspected mastoiditis. The plates in this case were poor, but they showed the bone pneumatic on both sides. There seems to be destruction of bone behind the right antrum. Mastoid operation was performed next day. Free pus and granulations were found, also perisinus abscess.

Case 1371.—M. 30. Dr. J. L. Adams. Double O. M. P. A. The plates showed the left mastoid generally diploetic throughout, except a few small cells above the antrum. About two-thirds down from the antrum is a large cell or abscess cavity, and a small one just above it. The sinus seemed fairly well forward, and not very deep. The right seemed mixed, finely cellulodiploetic throughout. The sinus could not be seen. Left simple mastoidectomy four days later. A large tip cell was found containing about a dram of pus. It was in contact with the sinus.

Case 1380.—F. 22. Dr. F. Whiting. Right O. M. P. A. Mastoiditis. The plates showed the mastoids of pneumatic type, the cells increasing in size downward and backward, the largest behind and below. The left showed sharp and clear. The right was cloudy. Will probably show a very large cell at the posterior lower margin of process and one overlapping posterior margin of sinus. The sinus seemed to be in about the usual position. Right simple mastoidectomy. Cortex soft. Large tip cell filled with pus. Perisinus abscess. Sinus covered with granulations. Small cells of tip, the zygoma and antrum filled with granulations.

Case 1474.—M. Dr. Bryant. Left O. M. P. A. Mastoiditis. Plates showed the right mastoid to be of rather small cell pneumatic type. Septa clear and distinct. The left was similar in type, but the septa were indistinct. The sinus was well forward, and the operator was asked to look for perisinus abscess above emissary vein. Operation was performed the same day. Tip cells were filled with pus. A large cell below led to epidural cerebellar abscess. The cells in zygoma contained pus. Small area of dura in the middle fossa and the sinus were exposed.

Fourth: Fractures of the mastoid process can be shown, but not invariably. Stereograms will aid in these cases.

Fifth: Faults in the canal wall and in the tegmen tympani and antri have, I believe, been frequently shown. We have shown two.

Sixth: Furunculosis of the canal gives marked symptoms of mastoid involvement in many instances. The diagnosis in the vast majority of these cases can be cleared up by an X-ray examination.

Case 1343.—M. 41. Dr. Yates. Left furunculosis, suspected mastoid. Skiagraph showed only a few cells about the mastoid antrum and along the anterior border of the sinus, on right side. It showed the left finely diploetic, a little hazy, but if diseased only slightly. No cavities. The furuncle was incised and no further operation was necessary.

Case 1347.—M. 22. Dr. Kenefick. Left furunculosis, O. M. P. A., and suspected mastoid. Mastoid was diploetic on both sides. No evidence of mastoiditis was shown by the plates. Sinus was seen to be moderately well forward. The furuncle was incised and no further operation was required.

Case 1348.—F. 19. Dr. Kenefick. Furunculosis, suspected right mastoiditis. Skiagraph showed a rather small cell pneumatic type of bone on both sides. The right antrum was a little hazy. Sinus fairly well forward. The furuncle was incised and no further operation required.

Case 1367.—M. 10. Dr. Lewis. Right furunculosis. O. M. P. A., suspected mastoiditis. Skiagraph showed both mastoids of pneumatic type, and the right side clear. Furuncle incised, no further operation required.

The conclusions which can be properly drawn from these cases are quite plain. It is a safe rule not to make a diagnosis on one point, and this applies to X-ray findings in the mastoid as much as to anything else. But such an examination of the mastoid process is of great value, and this statement cannot be successfully challenged. It is backed up by the experience of radiologists and surgeons all over the country, many of whom have been working in this line a great deal longer than I have. Ordinary common sense must be used in reading the plates, and the findings should be supported by the clinical symptoms. Where the clinical symptoms alone are not sufficient for a diagnosis of mastoiditis, a positive plate will frequently supply conclusive evidence for immediate operation, as in case 977 just cited.

CONCLUSIONS.

If our experience at the New York Ear and Ear Infirmary teaches anything, it is that every case of acute suppurative disease of the middle ear should be skiagraphed, as well as many of the furuncular cases. Skiagraphs would also be useful where the radical mastoid operation is to be performed. Anything which will add to our knowledge of a case will increase the chances of the patient's speedy recovery.

In conclusion, it is not to be understood that the blood count, the form of infection, or positive X-ray findings alone can be relied upon to any considerable extent as determining the necessity for a mastoid operation. The clinical symptoms are all important; without them our modern aids are of little value, for intestinal disturbances will occasionally cause a comparatively high leucocytosis and a rather alarming percentage of polynuclear cells. We may have a rather violent streptococcus infection in the canal, or a pneumococcus infection which looks vicious in the smear, but both may recover after myringotomy, though there may have been considerable mastoid tenderness. A streptococcus mucosæ otitis media may eventuate in the same manner. Not infrequently the clinical symptoms are not sufficient to warrant surgical interference in this form of infection. When the acute stage has passed we may have none, no pyrexia, no pain, no tenderness, only the otorrhea remaining, or it may also have ceased. The blood count is frequently normal. Wait for further clinical symptoms in these cases, and the chances are greatly in favor of their indicating meningitis or sinus thrombosis when they do arrive, instead of a simple acute mastoiditis. A positive X-ray plate in such a case, taken in connection with the form of infection, settles the diagnosis and makes the mastoid operation imperative with as little delay as possible.

In either streptococcus or pneumococcus cases (though we believe more liberty can be taken with the latter, as a rule) the danger signal may be a sudden rise in the polynuclear count, with or without an increased leucocytosis, especially the latter. A positive X-ray plate will again settle the diagnosis, though the clinical symptoms may not alone be sufficient to indicate the mastoid operation.

Staphylococcus infections seem to allow more latitude than any of the other forms of infection, but these will give us plenty of trouble if the drainage is poor. The same applies here as to the streptococcus form of infection.

Finally, a certain number of cases will occur in which symptoms of sinus thrombosis may be the first indication that the time for operative interference has arrived. A positive blood culture in such a case will, of course, settle the diagnosis, provided other sources of infection of the blood stream can be eliminated.

The differential blood count in these cases is variable and cannot be regarded as affording any very positive assistance in the diagnosis, but a low leucocyte count and a high polynuclear percentage is suspicious.

XX.

OCULAR MANIFESTATIONS IN NASAL AND AURAL DISEASES WHICH PROBABLY INDICATE INVOLVEMENT OF THE SYMPATHETIC NERVOUS SYSTEM.*

By W. H. Haskin, M. D.,

New York.

In recent years a great deal has been written about the ocular symptoms in both aural and nasal diseases, and below is given a brief summary of the many conditions which have been described by the various writers. It is unfortunate that there has been no definite system of differentiating the various symptoms, especially when considering the optic nerve. There is an absolute difference between optic neuritis and choked disc, and care should be taken not to describe the one when the other is really present. The writer has roughly grouped the numerous symptoms given, as follows:

1. Amblyopia, amaurosis, amaurotic amblyopia, scotomas, and contracted fields.
2. Discs: hyperemic, outlines pale and indistinct, congestion, margins of the nerve indistinct or hazy, with hyperemia of the disc.
3. Papillæ: white and atrophic without showing optic neuritis, prepapillary edema, papillitis.
4. Retina: retinitis, retinochoroiditis, edema of, detachment of, edema with exudate, swelling and tortuosity of the retinal vessels, vessels overfull, general edematous condition of the fundus, hyperemia of the arteries, pulsation of the arteries and veins.
5. Nerve: unilateral and bilateral neuritis, retrobulbar and perineuritis.

---

*Read before the American Laryngological, Rhinological and Otological Society, Washington, May 10, 1913.

6. This includes punctate keratitis, vitreous opacities, exophthalmos, increased intraocular tension, edema of the upper lids, dilatation of the pupil, oculomotor paralysis, abducens paralysis, disturbances of lacrimation, photophobia.

In addition to these symptoms, pain is generally present, although it varies greatly in character. Dr. Sluder has also noted disturbances of taste, and has noted paralyses of the palate muscles, which are supplied by the superior maxillary branch of the fifth nerve. Intractable sneezing, profuse rhinorrhea, a tickling sensation in the palate, and areas of anesthesia have also been described.

Dr. Wolff Freudenthal has reported a significant case of angioneurotic edema of the face occurring in a patient upon whom he had previously operated for frontal sinusitis. Five years before operation she had suffered for a year with a profuse hydrorrhea nasalis of the left nostril only, which had not responded to treatment, but finally disappeared of itself. This was undoubtedly a case of true involvement of the sympathetic.

The relationship of the optic nerve to the postethmoidal cells and to the sphenoidal sinuses has been very thoroughly described by many, but very few have apparently noticed the intimate association existing between the fifth nerve and the sphenoids. Dr. Sluder and Onodi have also noted the proximity of the other orbital nerves to the sphenoid sinuses, and the possibility of their involvement in disease of the sinuses. The writer has never found these relationships, with the exception of the second branch of the fifth nerve, which is very frequently and extensively in contact with the sinus, both the sensory portion and the motor and the sympathetic, i. e., the Vidian portion, lying on a portion of the sinus where they would be more likely to be involved, if the sinus were inflamed, than the optic nerve itself. The writer wishes to add the sympathetic nerves to the numerous relations already noted, and to emphasize the very intimate contact that exists between these nerves and the bony walls of the sinuses. As the majority of the symptoms (ocular) are due to vasomotor disturbances, and as the sympathetic nerves are the vasomotor nerves, it seems to the writer that we have a most reasonable explanation for most of these manifestations, in that they are caused by irritation of this system. An examination of the specimens will show how slightly any relationship can exist between the third, the fourth, the first branch of the fifth or the sixth nerves. They all enter the cavernous sinus external to the carotid artery (which latter extends the whole length of the sinus and lies on its inner wall), being covered by its endothelial lining membrane, except the sixth, which lies on the external wall of the artery. They all enter the orbit from this outer wall, passing through the common tendon of the orbital muscles, and the writer has yet to see a specimen showing any actual relationship of these nerves and the sinuses. With the second branch of the fifth, however, the relationship is very intimate,as said before, and the third branch is frequently in relation with the sinus, as can be seen on the specimens. Dr. Sluder even claims to have seen the Vidian nerve actually exposed in the sinus. The writer has never been satisfied with the various theories in explanation of the many ocular symptoms in these cases in which there are no intracranial complications, orbital abscesses, or even pressure within the diseased sinuses themselves. He has been unable to find any reports of microscopic proof of neuritis, except where meningitis was present. It is well recognized that increased intracranial pressure produces characteristic ocular disturbances, and optic neuritis, brain tumors, gummata, and subdural or brain abscesses are among the many things to be remembered and to be determined in our efforts to interpret the eye symptoms. The writer desires to speak only of those cases in which the ocular disturbances are relieved by treatment of the nasal sinuses. In order to fully appreciate the reasonability of the writer's views, a complete description of the anatomy and the physiology of these nerves is given below, being taken from Gray's anatomy and from Landois' physiology.

The third nerve anastomoses are: (a) On the cavernous sinus with the first branch of the fifth, receiving its sensory fibers supplying the iris; (b) with the sympathetic through the carotid plexus, and (c) indirectly with the sixth, whereby it receives its vasomotor fibers. It contains (1) voluntary motor fibers for all the orbital muscles except the external rectus and the superior oblique; (2) the fibers of the sphincter pupillæ, which are excited reflexly from the retina; (3) the fibers for the tensor choroideæ, which is the muscle of accommodation. The two latter sets form the short root of the ciliary ganglion, and reach the eyeball through the ciliary nerves. This nerve controls the pupillary action associated with accommodation. Stimulation of this nerve causes contraction of the pupil, while paralysis causes dilatation of the pupil. It should be remembered that in paralysis of the third nerve the pupil will not contract, either for light or for accommodation, whereas stimulation causes contraction.

The fourth nerve anastomoses with the carotid plexus and with the first branch of the fifth. It supplies the superior oblique muscle.

In describing the fifth nerve, it should be borne in mind that the origins of its sensory root anastomose with the motor nuclei of all the nerves arising from the medulla oblongata, except that of the sixth, as this fact explains the vast number of reflex relations of this nerve. As it crosses over the superior border of the petrous portion of the temporal bone, it is in direct contact with the superior petrosal sinus. It then becomes enlarged as the Gasserian ganglion, and lies directly over the carotid canal of the petrous bone and receives sympathetic fibers from the carotid plexus. Landois says that it also receives fibers from the cavernous plexus. The following description of the first and second branches is given in full from Landois, and should be carefully studied:

I. The ophthalmic division receives sympathetic fibers (vasomotor nerves) from the plexus cavernosus; it passes through the superior orbital fissure (sphenoidal) into the orbit. Its branches are:

1. The small recurrent nerve which gives sensory branches to the tentorium cerebelli. Fibers—the vasomotor nerves for the dura mater—proceed along with it from the carotid plexus of the sympathetic.

2. The lacrimal nerve gives off (a) sensory branches to the conjunctiva, the upper eyelid, and the neighboring part of the skin over the temple; (b) true sensory fibers to the lacrimal gland. Stimulation of this nerve is said to cause a secretion of tears, while its section prevents the reflex secretion excited through the sensory nerves of the eye. After a time section of the nerve is followed by a paralytic secretion of tears, although the statement is contested by Reich. The

secretion of tears may be excited reflexly, by strong stimulation of the retina by light, by stimulation of the first and second branches of the trigeminus, and through all the sensory cranial nerves.

3. The frontal (f) gives off the supratrochlear, which supplies sensory fibers to the upper eyelids, brow, glabella, and those which excite the secretion of tears reflexly; and by its supraorbital branch (b), analogous branches to the upper eyelid, skin of the forehead, and the adjoining skin over the temple as far as the vertex.

4. The nasociliary nerve (nc), by its infratrochlear branch supplies fibers, similar to those of 3, to the conjunctiva, caruncula and sacculus lacrimalis, the upper eyelid, brow, and root of the nose. Its ethmoidal branch supplies the tip and alæ of the nose, outside and inside, with sensory branches, as well as the upper part of the septum and the turbinated bones with sensory fibers, which can act as afferent nerves in the reflex secretion of tears; while it is probable that vasomotor fibers are supplied to these parts through the same channel. (These fibers may be derived from the anastomosis with the sympathetic.) The nasociliary nerve gives off the long root (1) of the ciliary (c), and 1 to 3 long ciliary nerves.

The ciliary ganglion which, according to Schwalbe, perhaps belongs rather to the third than the fifth nerve, has three roots—(a) the short or oculomotorius; (b) the long (L), from the nasociliary; and (c) the sympathetic (s) sometimes united with b, from the carotid plexus. The short ciliary nerves (t), six to ten in number, proceed from the ganglion, along the long ciliary nerves, to near the entrance of the optic nerve, where they perforate the sclerotic coat and run forwards between it and the choroid. Tiedemann says that it also communicates with Meckle's.

Ciliary Nerves.—Physiologically these nerves contain:

1. The motor fibers of the sphincter pupillæ and the tensor choroideæ from the root of the oculomotorius.

2. Sensory fibers of the cornea, which are distributed as excessively fine fibrils between the epithelium of the conjunctiva bulbi; they perforate the sclera. These fibers cause a reflex secretion of tears (N. lacrimalis) and closure of the eyelids (N. facialis). Sensory fibers are supplied to the iris (pain in iritis and operations on the iris), the choroid (pain-

ful tension when the ciliary muscle is strained), and the sclerotic.

3. Vasomotor nerves for the blood vessels of the iris, choroid, and retina. They arise in part from the sympathetic root, and the anastomosis of the sympathetic with the ophthalmic division of the trigeminus. The iris and retina receive most of their vasomotor nerves from the trigeminus itself, and a few from the sympathetic direct.

4. Motor fibers for the dilator pupillæ, which for the most part are derived from the sympathetic, through the sympathetic root of the ganglion and the anastomosis of the sympathetic with the trigeminus. Some observers deny altogether the existence of a dilator pupillæ muscle. The ophthalmic division contains independent fibers for the dilatation of the pupil, which arise in the medulla oblongata and proceed directly into the ophthalmic or arise from the Gasserian ganglion.

After section of the trigeminus the pupil becomes contracted after a short period of dilatation (rabbit, frog), but this effect is not permanent. After excision of the superior cervical ganglion of the sympathetic, the power of dilatation of the pupil is not completely abolished. The narrowing of the pupil which follows section of the trigeminus in the rabbit, and which rarely lasts more than half an hour, may be regarded as due to a reflex stimulation of the oculomotorius fibers of the sphincter, in consequence of the painful stimulation caused by section of the trigeminus.

Stimulation of the Cervical Sympathetic.—Either in the neck or in its course to the eye, when the peripheral end of the cervical sympathetic is stimulated, besides causing constriction of the blood vessels on that side of the head, there is dilatation of the pupil, as well as contraction of the smooth muscular fibers (Müller's muscle) in the orbit and eyelids. The membrane of the inferior orbital fissure (sphenomaxillary fissure) in man has a layer of smooth muscle, one millimeter thick, and arranged for the most part longitudinally. Both eyelids contain smooth muscular fibers which serve to close them; in the upper lid they lie close under the conjunctiva. Tenon's capsule also contains smooth muscular fibers. The sympathetic nerve supplies all these muscles (the orbital muscle is partly supplied from the sphenopalatine ganglion). Hence stimulation of the sympathetic causes dilatation of the pupil and of the palpebral fissure, with protusion of the eyeball. This result may be caused reflexly by strong stimulation of sensory nerves. Strong stimulation of the nerves of the sexual organs is followed by similar phenomena in the eye. The dilatation of the pupil which occurs in children affected with intestinal worms is perhaps an analogous phenomenon. The pupil is dilated when the spinal cord is stimulated (at the origin of the sympathetic), as in tetanus.

Section of the cervical sympathetic, besides other effects, notably those on the blood vessels on that side of the head and face, causes narrowing of the fissure between the eyelids and the eyeball sinks in its socket.

5. It is probable that trophic fibers occur in the trigeminus and pass through the ciliary nerves to reach the eye. If the trigeminus be divided within the cranium, after six to eight days, inflammation, necrosis of the cornea, and ultimately complete destruction of the eyeball take place, constituting panophthalmia.

Section of the trigeminus paralyzes the vasomotor nerves in the interior of the eyeball, which must undoubtedly cause a disturbance in the intraocular circulation. According to Jesner and Grünhagen, the trigeminus also contains vasodilator fibers, whose stimulation is followed by increased flow of blood to the eye, with consecutive excretion of the fibrin factors and increase in the amount of albumin of the aqueous humor. After section of the nerve the intraocular tension is diminished, while stimulation of the nerve is followed by increase of the intraocular pressure. This diminution of the normal tension necessarily must alter the normal relation of the filling of the blood and lymph vessels, and also the movement of the fluids upon which the normal nutrition is largely dependent.

There are three conditions on which the above changes may depend: (1) Mere loss of sensibility, which alone is not sufficient to explain the phenomena; (2) vasomotor disturbance, which is excluded by the above facts, and also by the other consideration that if the fifth nerve be divided and the superior cervical ganglion excised simultaneously, ophthalmia does not occur, and, in fact, excision of this sympathetic ganglion may modify the results of section of the fifth.

Thus we are forced to (3) the theory of trophic fibers, whose center is the Gasserian ganglion.

Pathologic.—In cases of anesthesia of the trigeminus in man, and, more rarely, in severe irritation of this nerve, inflammation of the conjunctiva, ulceration and perforation of the cornea, and finally panophthalmia, have been observed. This condition has been called ophthalmia neuroparalytica.

There are other affections of the eye depending upon disease of the vasomotor nerves, which are quite different from the foregoing, as they never lead to degenerative changes: such as ophthalmia intermittens (due to malaria); a unilateral, intermittent, excessive filling of the blood vessels of the eye, accompanied by the secretion of tears; photophobia, often accompanied by iritis, and effusion of pus into the chambers of the eye. This condition is regarded by Eulenburg as a vasoneurotic affection of the ocular blood vessels. Pathologic observations, as well as experiments upon animals, have shown that there is an intimate physiologic connection between the vascular areas of both eyes, so that affections of the vascular area of one eye are apt to induce similar disturbances of the opposite eye. This serves to explain the fact that inflammatory processes in the interior of one eyeball are apt to produce a similar condition in the other eye. This is the socalled "sympathetic ophthalmia." It is probably effected through Ribes's ganglion. Thus, stimulation of the ciliary nerves or the fifth on one side causes dilatation of the blood vessels, not only on its own side, but also on the other side as well. The pathologic condition of glaucoma simplex, where the intraocular tension is greatly increased, is ascribed by Donders to irritation of the trigeminus. (Increased intraocular tension may be produced by irritation of the secretory fibers contained in the fifth nerve, by stimulating the nucleus of the trigeminus in the medulla oblongata, and also reflexly by irritation of the peripheral branches of the fifth, as by nicotin placed in the eye. It is possible, however, that some forms of glaucoma are produced by diminished removal of the aqueous humor from the eye.) Unilateral secretion of tears, due to irritation of the ophthalmic division of the fifth, has been repeatedly observed, but unilateral cessation of tears, due to paralytic conditions, very rarely.

II.—Superior Maxillary Division.—It gives off

1. The delicate recurrent nerve, a sensory branch to the dura mater, which accompanies the vasomotor nerves, derived from the superior cervical ganglion of the sympathetic, and is distributed to the area of the middle meningeal artery.

2. The subcutaneous malar or orbital (o) supplies by its temporal and orbital branches sensibility to the lateral angle of the eye and the adjoining area of skin of the temple and cheek. Certain fibers are said to be the true secretory nerves for tears.

3. The dental, anterior, posterior, and median, and with them the anterior fibers from the infraorbital nerve, supply sensory fibers to the teeth in the upper jaw, the gum, periosteum, and the cavities of the jaw. The vasomotor nerves of all these parts are supplied from the upper cervical ganglion of the sympathetic.

The sphenopalatine ganglion (Meckel's — n) forms connections with the second division. To it pass two short sensory root fibers from the second division itself, which are called sphenopalatine. Motor fibers enter the ganglion from behind, through the large superficial petrosal branch of the facial (j); and gray vasomotor fibers (v) from the sympathetic plexus on the carotid (the deep large petrosal nerve). The motor and vasomotor fibers from the Vidian nerve, which reach the ganglion through the canal of the same name.

Branches of Meckel's Ganglion.—(1) The sensory fibers (N) which supply the roof, lateral walls and septum of the nose (posterior and superior nasal); the terminal fibers of the nasopalatine pass through the canalis incisivus to the hard palate, behind the incisor teeth. The sensory inferior and posterior nasals for the lower and middle turbinate bones, and both lower nasal ducts, are derived from the anterior palatine branch of the ganglion, which descends in the palatomaxillary canal. (See illustrations I and III.) Lastly, the sensory branches of the hard (p) and soft palate (pl) and the tonsils arise from the posterior palatine nerve. All the sensory fibers of the nose (see also the ethmoidal nerve), when stimulated, cause the reflex act of sneezing. Preparatory to the act of sneezing there is always a peculiar feeling of tickling in the nose, which is perhaps due to dilatation of the nasal blood vessels. This dilatation is rapidly caused by cold, more especially when it is applied directly to the skin. The dilatation of the vessels is followed by an increased secretion of watery fluid from the nasal mucous membrane. Stimulation of the nasal nerves also causes a reflex secretion of tears, and it may also cause a standstill of the respiratory movements of the expiratory phase.

(2) The motor branches descend in the posterior palatine nerve through the small palatine canal, and give off (h) motor branches to the elevator of the soft palate and azygos uvulæ. The sensory fibers of these muscles are supplied by the trigeminus. According to Politzer, spasmodic contraction of these muscles occasionally causes crackling noises in the ears.

(3) The vasomotor nerves of this entire area arise from the sympathetic root, i. e., from the upper cervical ganglion. The root of the trigeminus supplies the secretory nerves of the mucous glands of the nasal mucous membrane. Stimulation excites the secretion, while section of the trigeminus decreases it with simultaneous atrophic degeneration of the mucous membrane. Thus, trophic functions for the mucosa have been ascribed to the trigeminus.

The third branch is rarely involved in the conditions we are studying, supplying as it does the lower part of the face and neck. It is closely associated, however, with the other branches through its ganglion and through the tympanic plexus, which accounts for the pains which are often reflected to the ear when the fifth nerve is really the site of the lesion itself. Below is given the anatomy of the cephalic portion of the sympathetic, being taken from Gray's anatomy:

THE SYMPATHETIC NERVE.

The sympathetic nerve is socalled from the opinion entertained that through it is produced a sympathy between the affections of distant organs. It consists of a series of ganglia, connected together by intervening cords, extending on each side of the vertebral column from the base of the skull to the coccyx. It may, moreover, be traced up into the head, where the ganglia (which are all in connection with the fifth cranial nerve) occupy spaces between the cranial and facial bones.

The sympathetic nerve, for convenience of description, may be divided into several parts, according to the position occupied by each. Of these we will briefly describe:

1. Cephalic portion ....4 pairs of ganglia
2. Cervical portion ....3 pairs of ganglia

Each ganglion may be regarded as a distinct center, from or to which branches pass in various directions. These branches may be thus arranged: 1. Branches of communication between the ganglia. 2. Branches of communication with the cerebral or spinal nerves. 3. Primary branches passing to be distributed to the arteries in the vicinity of the ganglia, and to the viscera, or proceeding to other ganglia placed in the thorax, abdomen or pelvis.

Of these the primary branches of distribution also consist of two kinds of nerve fibers, the sympathetic and the spinal. They have a remarkable tendency to form intricate plexuses, which encircle the blood vessels, and are conducted by them to the viscera.

The cephalic portion of the sympathetic consists of four pairs of ganglia: 1. The ophthalmic ganglion. 2. The sphenopalatine, or Meckel's ganglion. 3. The otic, or Arnold's ganglion. 4. The submaxillary ganglion. These have been already described in connection with the three divisions of the fifth nerve. There is also the single ganglion uniting the two sides, i. e., Ribes' ganglion.

CERVICAL PORTION OF THE SYMPATHETIC.

The cervical portion of the sympathetic consists of three ganglia on each side, which are distinguished, according to their position, as the superior, middle, and inferior cervical. (See illustration No. I.)

The superior cervical ganglion, the largest of the three, and the one which is of interest to us, is placed opposite the second and third cervical vertebræ, and sometimes as low as the fourth or fifth. It is of a reddish gray color, and usually fusiform in shape; sometimes broad and occasionally constricted at intervals, so as to give rise to the opinion that it consists of the coalescence of several smaller ganglia. It is in relation, in front, with the sheath of the internal carotid artery and internal jugular vein; behind, it lies on the rectus capitus anticus major muscle.

Its branches may be divided into superior, inferior, external, internal and anterior.

The superior branch appears to be a direct continuation of the ganglion. It is soft in texture and of a reddish color. It ascends by the side of the internal carotid artery, and entering the carotid canal in the temporal bone, divides into two branches, which lie, one on the outer, and the other on the inner side of that vessel.

The outer branch, the larger of the two, distributes filaments to the internal carotid artery, and forms the carotid plexus.

The inner branch also distributes filaments to the internal carotid, and, continuing onwards, forms the cavernous plexus.

CAROTID PLEXUS.

The carotid plexus is situated on the outer side of the internal carotid. Filaments from this plexus occasionally form a small gangliform swelling on the under surface of the artery, which is called the carotid ganglion. The carotid plexus communicates with the Gasserian ganglion, with the sixth nerve, and sphenopalatine ganglion, and distributes filaments to the wall of the carotid artery, and to the dura mater (Valentin), while in the carotid canal it communicates with Jacobson's nerve, the tympanic branch of the glossopharyngeal.

The communicating branches with the sixth nerve consist of one or two filaments which join that nerve as it lies upon the outer side of the internal carotid. Other filaments are also connected with the Gasserian ganglion. The communication with the sphenopalatine ganglion is effected by the carotid portion of the Vidian nerve, which passes forward, through the cartilaginous substance filling the foramen lacerum medium, along the pterygoid or Vidian canal, to the sphenopalataine ganglion. In this canal it joins the petrosal branch of the facial, thus forming the Vidian.

CAVERNOUS PLEXUS.

The cavernous plexus is situated below and internal to that part of the internal carotid which is placed by the side of the sella turcica in the cavernous sinus, and is formed chiefly by the internal division of the ascending branch from the superior cervical ganglion. It communicates with the third, fourth, fifth and sixth nerves, and with the ophthalmic ganglion, and distributes filaments to the wall of the internal carotid. The branch of communication with third nerve joins it at its point of division; the branch to the fourth nerve joins it as it lies on the outer wall of the cavernous sinus; other filaments are connected with the under surface of the trunk of the ophthalmic nerve; and a second filament of communication joins the sixth nerve.

The filament of connection with the ophthalmic ganglion arises from the anterior part of the cavernous plexus; it accompanies the nasal nerve, or continues forwards as a separate branch.

The terminal filaments from the carotid and cavernous plexuses are prolonged along the internal carotid, forming plexuses which entwine round the cerebral and ophthalmic arteries; along the former vessel they may be traced on to the pia mater; along the latter, into the orbit, where they accompany each of the subdivisions of the vessel, a separate plexus passing from the arteria centralis retinæ into the interior of the eyeball. The filaments prolonged onto the anterior communicating artery form a small ganglion, the ganglion of Ribes, which serves, as mentioned above, to connect the sympathetic nerves of the right and left sides.

If we now take up the physiology of the sympathetic, it at once becomes interesting in connection with our subject. The functions attributed to the cervical portion are as follows:

1. Pupil dilating fibers. Section of the cervical sympathetic causes contraction, while irritation causes dilatation of the pupil.
2. Motor fibers for Müller's smooth muscle of the orbit.
3. Vasomotor fibers for the outer ear and side of the face, the tympanum, conjunctiva, iris, choroid and the retina in part.
4. Trophic fibers, sweat fibers for the face, and secretory fibers for the various glands, such as the lacrimal, the several salivary and the mucous glands.

With this sketch before us, the writer desires to read some extracts from a reprint recently sent to him by Dr. Gruening, after having discussed the question with him. In it he says that "at times the statement is made that an optic neuritis has developed into a choked disc, thus implying that choked disc is a more advanced stage of optic neuritis." This, he says, is an erroneous view. He describes choked disc as having an initial, an intermediate and an advanced stage, every one of which is the ophthalmoscopically visible expression of increased intracranial pressure. He then tells us why it is that when an optic neuritis is present there is never any choked disc, because the intervaginal spaces are sealed by exudative material so that the serum does not enter the lymph spaces of the optic nerve and the perilymphatic sheaths of the optic and retinal vessels. He then asks this pertinent question: "How can the choking of the disc, which is so far from the area of the disease, in purulent middle ear disease, be explained?" He acknowledges in answer to this question that there is no succinct and lucid explanation of this interrelation. He then asks: "But what causes the excess of the cerebrospinal fluid?" and in answer says that the theory of chemotaxis, as defined by Theodor Leber, may furnish the clue. That theory is a most difficult one to understand, and, to the writer, cannot explain why one orbit is so often affected and not both. Dr. Gruening also writes that another point of interest is that the degree of swelling may vary in the two discs. As we assume that the hydrostatic pressure is equal at all points within the brain cavity, there must be structural conditions causing these variations. In conclusion he says "that further confirmation that we have not to deal with inflammatory action and destruction of the nerve tissue in choked discs in otitis cases is supplied by the fact that on removal of the purulent area the optic nerve returns to the normal." When such an authority as Dr. Gruening offers such a paper before the International Otological Congress, it seems to be pretty conclusive evidence that as yet but little is known of the true conditions which are manifested in the eyes, and affords the writer some excuse for the length of the present paper. He asks that you bear well in mind, first, that the sympathetic nerve is the vasomotor nerve of the eye, that is, it controls the blood supply. Second, that these nerves, after leaving the superior cervical ganglion, form the carotid plexus, and then on entering the cavernous sinus form the cavernous plexus and lie on its inner wall in contact with the sphenoids. Third, that the branches, after leaving these plexuses, often run through small bony canals in the walls of cavities or sinuses that become inflamed, such as the Vidian, the greater and lesser petrosal nerves and the branches entering the tympanum. Remembering the above relations, and considering that almost all of the ocular disturbances are true disturbances of the vasomotor system, it seems to the writer that the rational explanation of these symptoms lies in correctly attributing them to the sympathetic nerve. The delicate fibers of this system are much more exposed to irritation from inflammations in the accessory sinuses and even in the ear than any of the other nerves, and reference to the physiology will show that any or all of these ocular symptoms have actually been produced by experiments on these nerves. Dr. J. D. Richards describes two cases of pupil dilatation following extensive exenteration of the petrous bone with exposure of the carotid artery in its canal, and attributed the dilatation to irritation of the plexus. In both cases, although the dilatation was very marked, still there was reaction to light and to accommodation, showing that the disturbance came through the fifth nerve. Although all writers maintain that choked disc is always caused by increased intracranial pressure, in most of our cases there are absolutely no other symptoms of this condition present, and it is certainly remarkable that in most cases the eye conditions clear up as soon as the local nasal or aural disease is cured or the irritation removed. Again, it is a very significant fact that in many cases where operation has cured the nasal disease and the eye symptoms have been relieved, an acute exacerbation will often be accompanied by a return of the eye symptoms, which will again disappear with the cure of the attack. True paralyses of the orbital muscles are not common and are more difficult to explain. However, the intimate relation of the sixth nerve, as it pierces the dura, with the two petrosal sinuses and then with the interior of the cavernous sinus itself, accounts for its implication whenever these sinuses are thrombosed.

Dr. Sluder has written often concerning the sphenopalatine ganglion and his method of treating it. He has undoubtedly been very successful, but an examination of the specimens presented will show how difficult it is to reach this region, and only an unusually skillful man with an absolute appreciation of the anotomy had better attempt these injections: It has required much time and great care for the writer to open the specimens in order to actually show the ganglion in its place where it is enmeshed in many blood vessels.

Let us look at the disease called glaucoma. This disease consists of a great increase in the intraocular tension (due to vasomotor disturbances), and frequently destroys the eye if not promptly relieved. Dr. Sluder reports ten cases of glaucoma in which he has relieved the pain by cocainization of the sphenopalatine foramen. Lapersonne and Fisch have reported cases of increased intraocular pressure caused b accessory sinus disease. Now turn back to the section on physiology and, under the ophthalmic branch of the fifth nerve, you will find that Donders has ascribed the greatly increased intraocular tension to irritation of the trigeminus. Read also in the same paragraph what Eulenberg says about ophthalmia intermittens, which he describes as an angioneurotic affection of the ocular blood vessels. This paragraph, in conclusion, says that "there are other diseases of the eye depending upon disease of the vasomotor nerves which are peculiar in that they do not lead to degenerative changes." Amaurosis, i. e., weakness of vision, and amblyopia frequently result from optic neuritis, but Landois says that both may be the signs of disturbances in other organs; that they may be sympathetic signs due to changes in the movement of the blood stream, depending upon stimulation of the vasomotor nerves. In fact, a careful study of the physiologic experiments upon the orbital nerves, and especially the sympathetic branches, will disclose the fact that all of the ocular manifestations that we see and have been so frequently described have been caused by section of or by irritation of the sympathetic nerve supply. There is one more point the writer in closing wishes to suggest, and that is the probable influence of the trophic nerve fibers. These are also sympathetic fibers conveying impulses from the centers to the periphery. Their end organs lie in the tissues themselves, although little is known about them. They are called trophic because they are supposed to govern or control the normal metabolism of the tissues. There are many diseases attributed to disease of these fibers or the centers from which they spring, such as the various forms of herpes, the condition known as "glossy skin," progressive muscular atrophy, myxoderma (seen in the nose as polypi), leucoderma, Addison's disease, anesthetic leprosy, and acute decubitus or bed sores. All nerves are supplied with trophic fibers, and it naturally follows that if these nerves become diseased and are destroyed, the nerve supplied by them will also atrophy and lose its function. To the writer this supplies a reasonable theory to explain why in cases of long standing disease of the sinuses, the disturbances of vision do not recover after the sinus disease has been cured, although in recent cases vision almost invariably does return. Irritation of any tissue for a short period of time will not produce any actual tissue change, but long continued irritation will gradually produce actual destruction, and that is what probably does take place in these eye cases. At first the sympathetic nerves are merely irritated by the inflammatory sinus and produce the symptoms of increased vasomotor action, but after long irritation the trophic nerves become destroyed and thus destroy the trophic control of the optic, and it loses its function, although an actual neuritis has never been present. When we consider that the majority of nasal affections are affections of the vasomotor nerves, and then realize that these same vasomotor branches are so intimately associated with the sphenoid sinuses, even before they join their individual ganglia, it does not seem to be unreasonable to advance the theories advanced in this paper, and the writer hopes that it will help all in the true interpretation of cases to be seen.

The writer desires to thank J. B. Lippincott & Co. and P. Blakiston's Son & Co. for their very kind permission to use the illustrations in this article, which are so much better than he could have prepared himself.

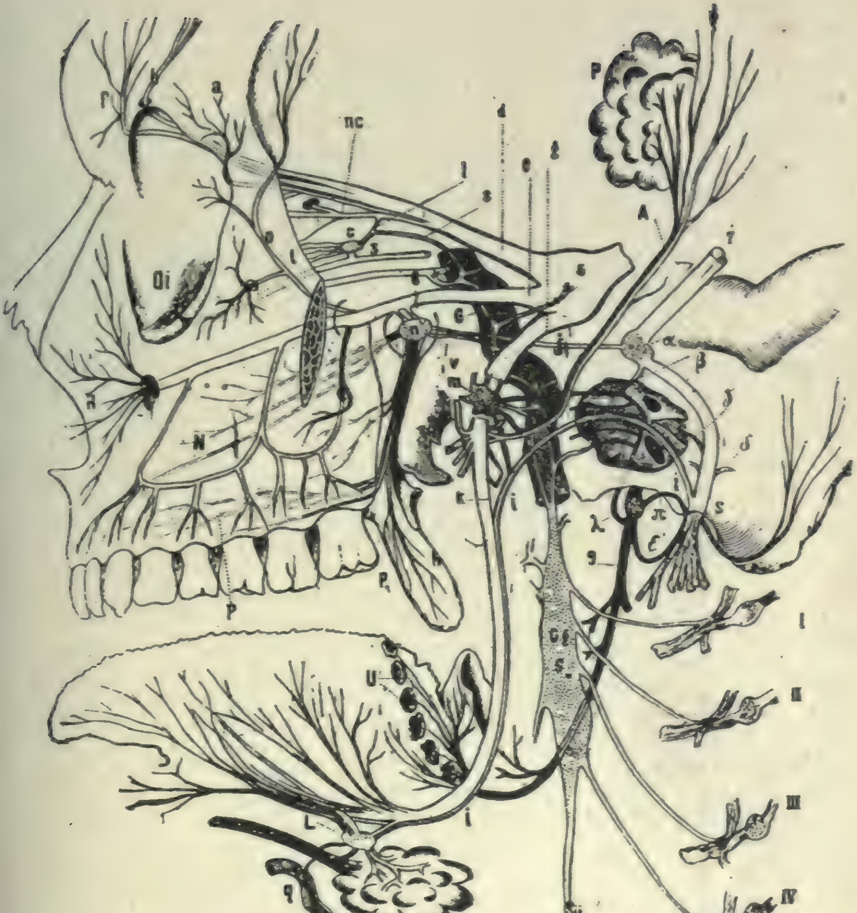
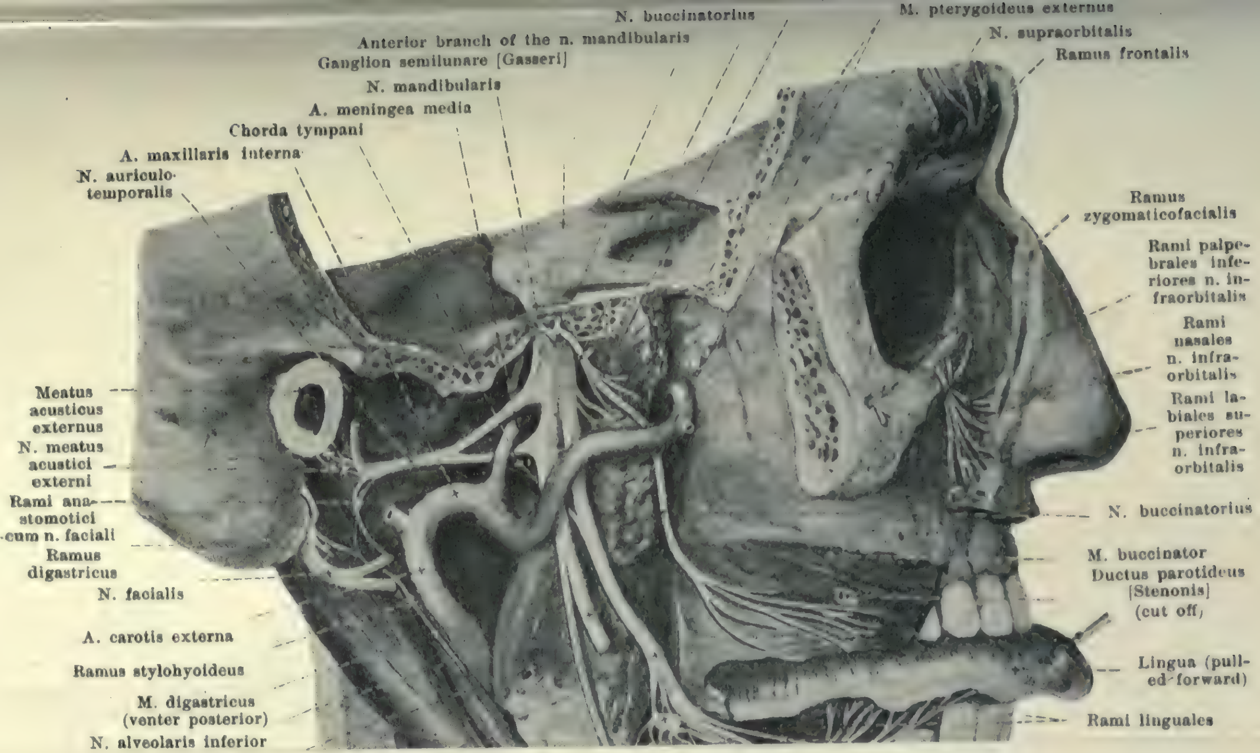

r
a
nc
d
1
2
6
8
P
A
7
c
3
Oi
4
5
G
n
v
m
R
N
k
i
α
β
γ
δ
s
λ
π
ε
9
P
p
I
II
III
IV
U
cg
L
q
M

FIGURE I.


N. buccinatorius
M. pterygoideus externus
Anterior branch of the n. mandibularis
N. supraorbitalis
Ganglion semilunare [Gasseri]
Ramus frontalis
N. mandibularis
A. meningea media
Chorda tympani
A. maxillaris interna
N. auriculo-temporalis
Ramus zygomaticofacialis
Rami palpebrales inferiores n. infraorbitalis
Rami nasales n. infraorbitalis
Meatus acusticus externus
Rami labiales superiores n. infraorbitalis
N. meatus acustici externi
Rami anastomotici cum n. faciali
N. buccinatorius
Ramus digastricus
M. buccinator
Ductus parotideus [Stenonis] (cut off)
N. facialis
A. carotis externa
Lingua (pulled forward)
Ramus stylohyoideus
M. digastricus (venter posterior)
Rami linguales
N. alveolaris inferior

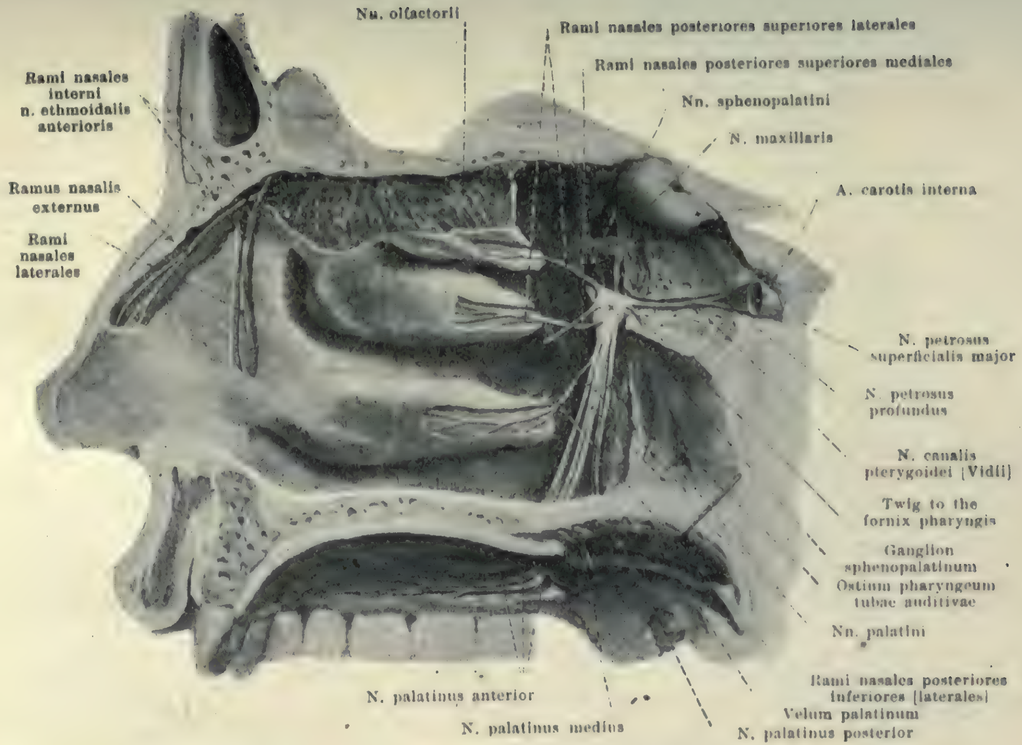

Nn. olfactorii
Rami nasales posteriores superiores laterales
Rami nasales posteriores superiores mediales
Rami nasales interni n. ethmoidalis anterioris
Nn. sphenopalatini
N. maxillaris
Ramus nasalis externus
A. carotis interna
Rami nasales laterales
N. petrosus superficialis major
N. petrosus profundus
N. canalis pterygoidei [Vidii]
Twig to the fornix pharyngis
Ganglion sphenopalatinum
Ostium pharyngeum tubae auditivae
Nn. palatini
Rami nasales posteriores inferiores [laterales]
N. palatinus anterior
Velum palatinum
N. palatinus medius
N. palatinus posterior

FIGURE III.

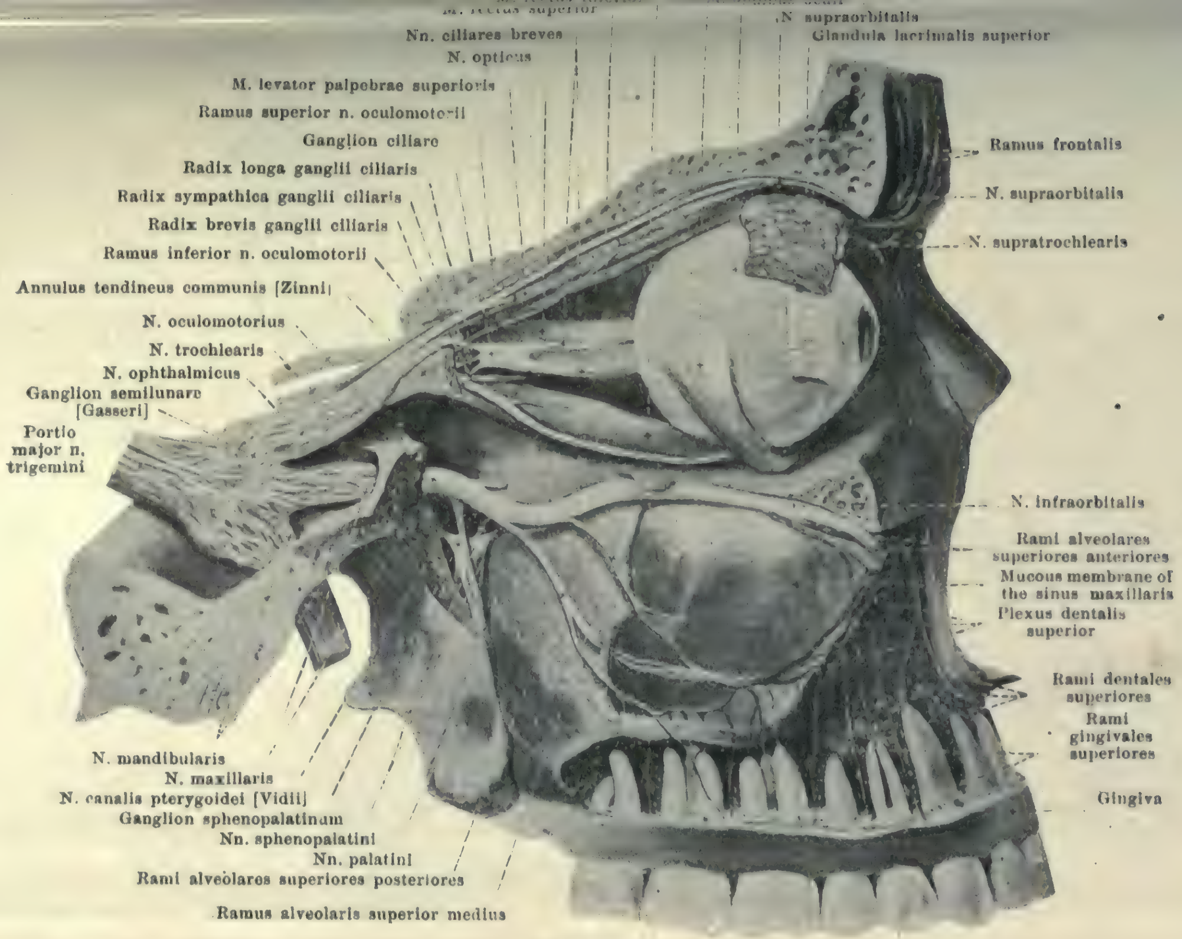

M. rectus superior
N supraorbitalis
Glandula lacrimalis superior
Nn. ciliares breves
N. opticus
M. levator palpebrae superioris
Ramus superior n. oculomotorii
Ganglion ciliare
Radix longa ganglii ciliaris
Radix sympathica ganglii ciliaris
Radix brevis ganglii ciliaris
Ramus inferior n. oculomotorii
Annulus tendineus communis [Zinni]
N. oculomotorius
N. trochlearis
N. ophthalmicus
Ganglion semilunare [Gasseri]
Portio major n. trigemini
Ramus frontalis
N. supraorbitalis
N. supratrochlearis
N. infraorbitalis
Rami alveolares superiores anteriores
Mucous membrane of the sinus maxillaris
Plexus dentalis superior
Rami dentales superiores
Rami gingivales superiores
Gingiva
N. mandibularis
N. maxillaris
N. canalis pterygoidei [Vidii]
Ganglion sphenopalatinum
Nn. sphenopalatini
Nn. palatini
Rami alveolares superiores posteriores
Ramus alveolaris superior medius

FIGURE IV.

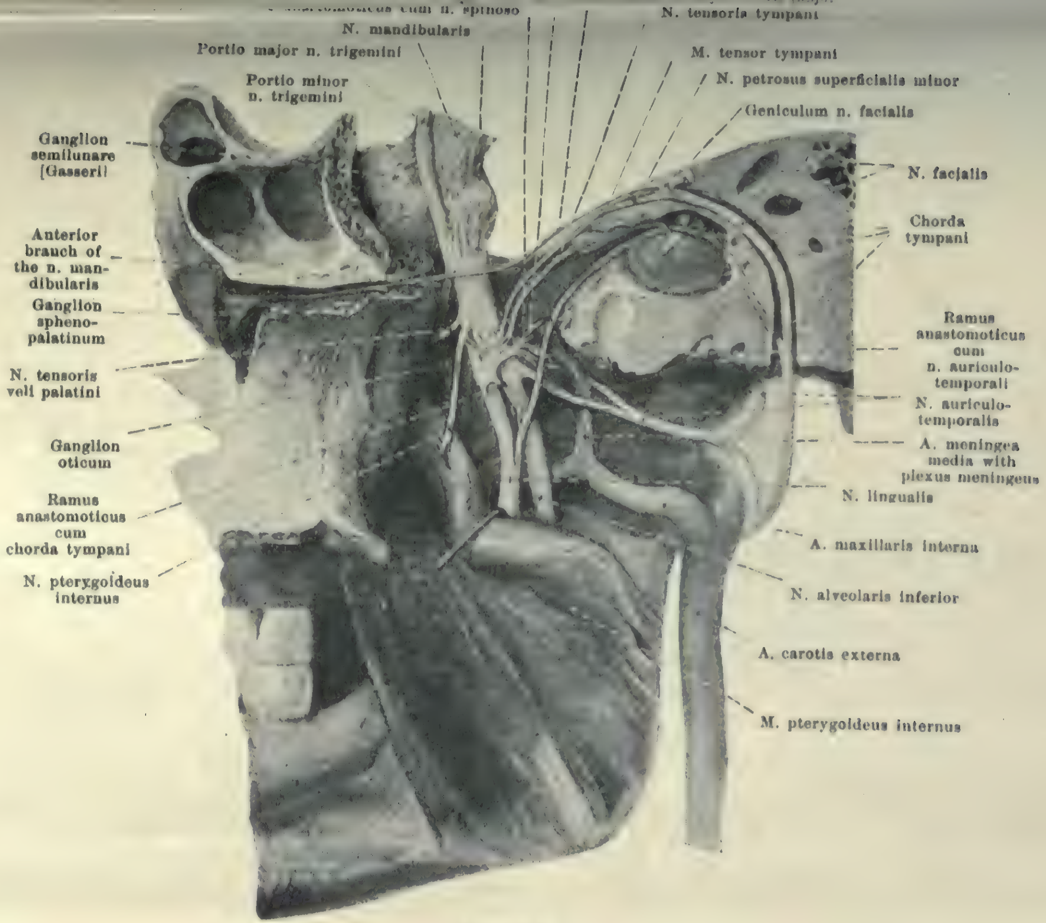
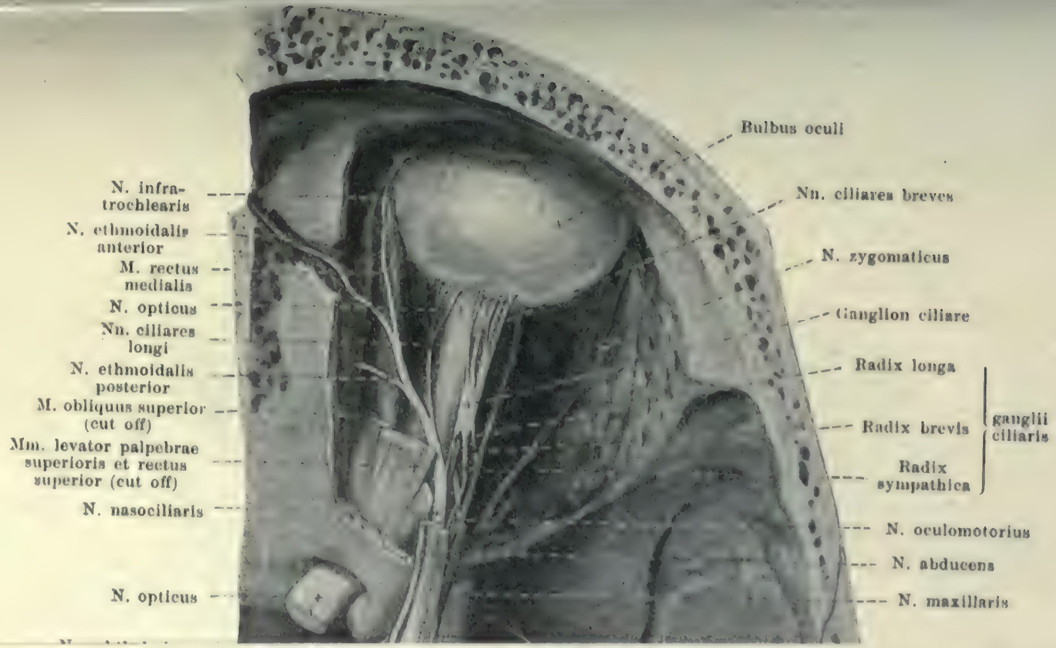

cum n. spinoso
N. mandibularis
Portio major n. trigemini
Portio minor n. trigemini
N. tensoris tympani
M. tensor tympani
N. petrosus superficialis minor
Geniculum n. facialis
Ganglion semilunare [Gasseri]
N. facialis
Chorda tympani
Anterior branch of the n. mandibularis
Ganglion spheno-palatinum
Ramus anastomoticus cum n. auriculo-temporali
N. tensoris veli palatini
N. auriculo-temporalis
Ganglion oticum
A. meningea media with plexus meningeus
N. lingualis
Ramus anastomoticus cum chorda tympani
A. maxillaris interna
N. pterygoideus internus
N. alveolaris inferior
A. carotis externa
M. pterygoideus internus

FIGURE V.



N. glossopharyngeus

FIGURE VII.

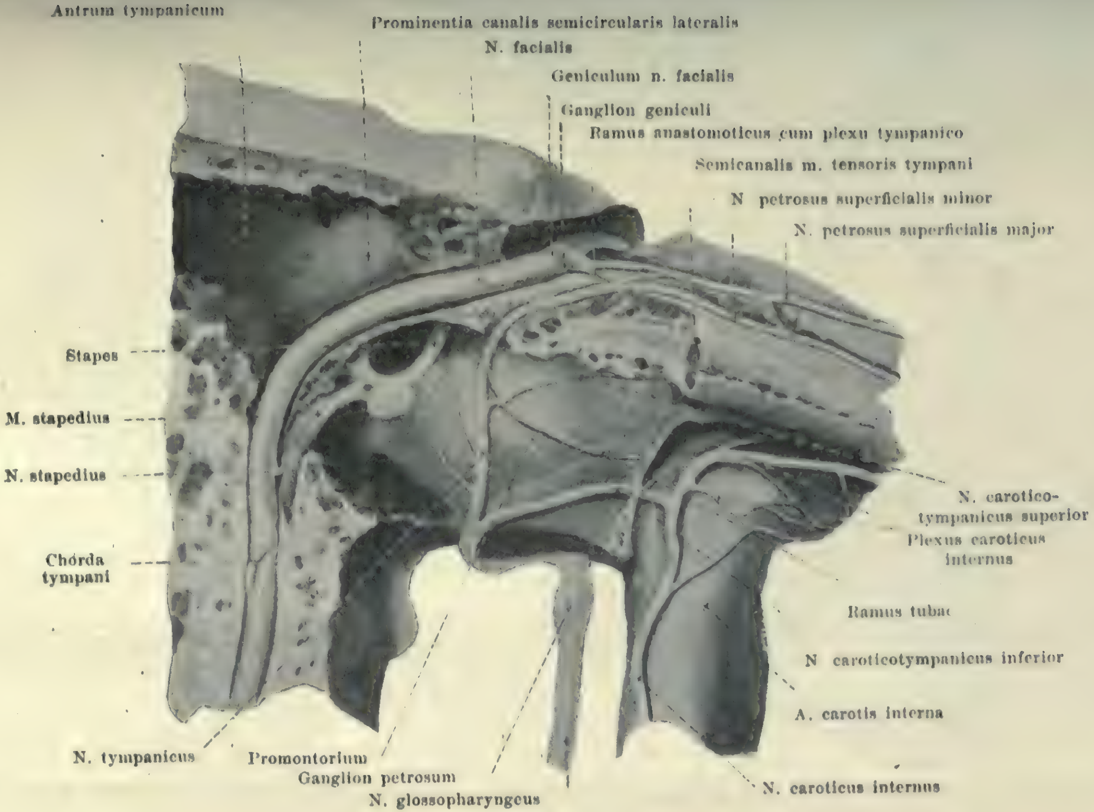

Antrum tympanicum
Prominentia canalis semicircularis lateralis
N. facialis
Geniculum n. facialis
Ganglion geniculi
Ramus anastomoticus cum plexu tympanico
Semicanalis m. tensoris tympani
N petrosus superficialis minor
N. petrosus superficialis major
Stapes
M. stapedius
N. stapedius
Chorda tympani
N. carotico-tympanicus superior
Plexus caroticus internus
Ramus tubae
N caroticotympanicus inferior
A. carotis interna
N. tympanicus
Promontorium
Ganglion petrosum
N. glossopharyngeus
N. caroticus internus

FIGURE VII.

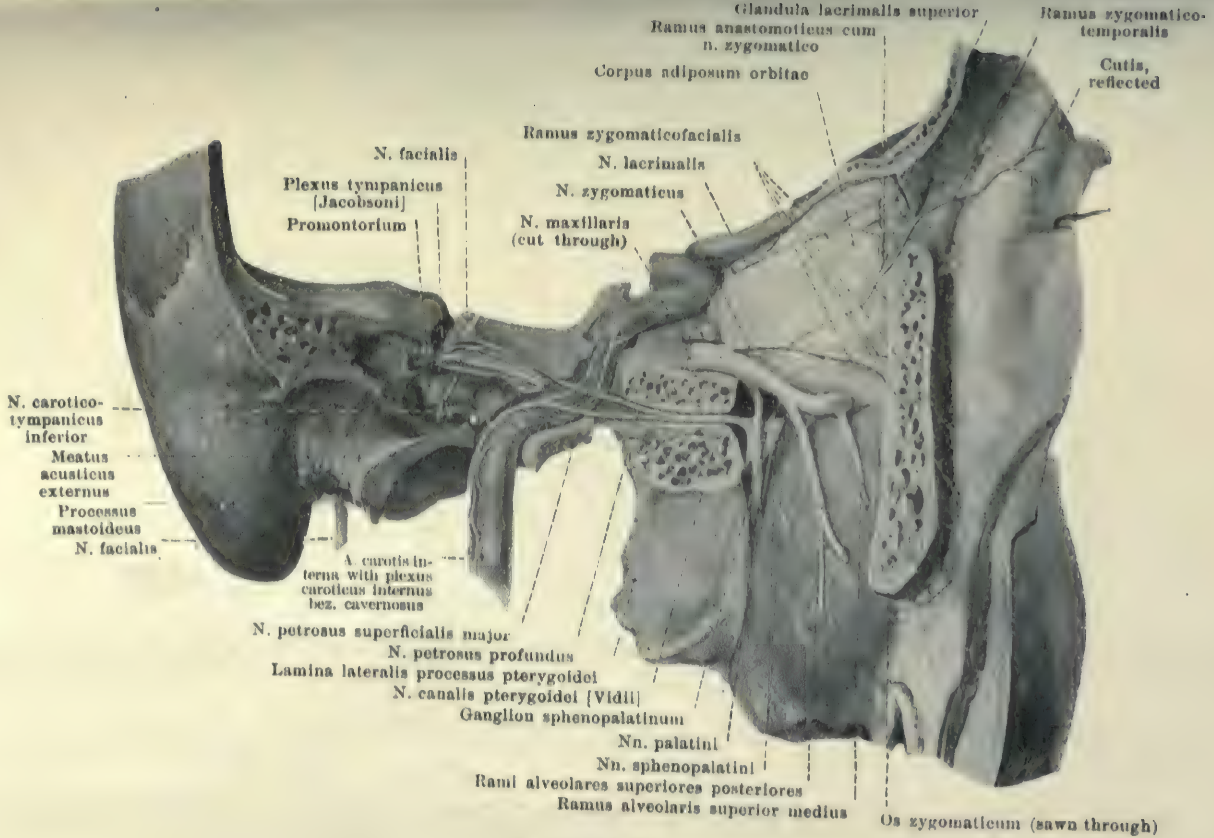

Glandula lacrimalis superior
Ramus zygomaticotemporalis
Ramus anastomoticus cum n. zygomatico
Cutis, reflected
Corpus adiposum orbitae
Ramus zygomaticofacialis
N. facialis
N. lacrimalis
Plexus tympanicus [Jacobsoni]
N. zygomaticus
Promontorium
N. maxillaris (cut through)
N. caroticotympanicus inferior
Meatus acusticus externus
Processus mastoideus
N. facialis
A. carotis interna with plexus caroticus internus bez. cavernosus
N. petrosus superficialis major
N. petrosus profundus
Lamina lateralis processus pterygoidei
N. canalis pterygoidei [Vidii]
Ganglion sphenopalatinum
Nn. palatini
Nn. sphenopalatini
Rami alveolares superiores posteriores
Ramus alveolaris superior medius
Os zygomaticum (sawn through)

FIGURE VIII.

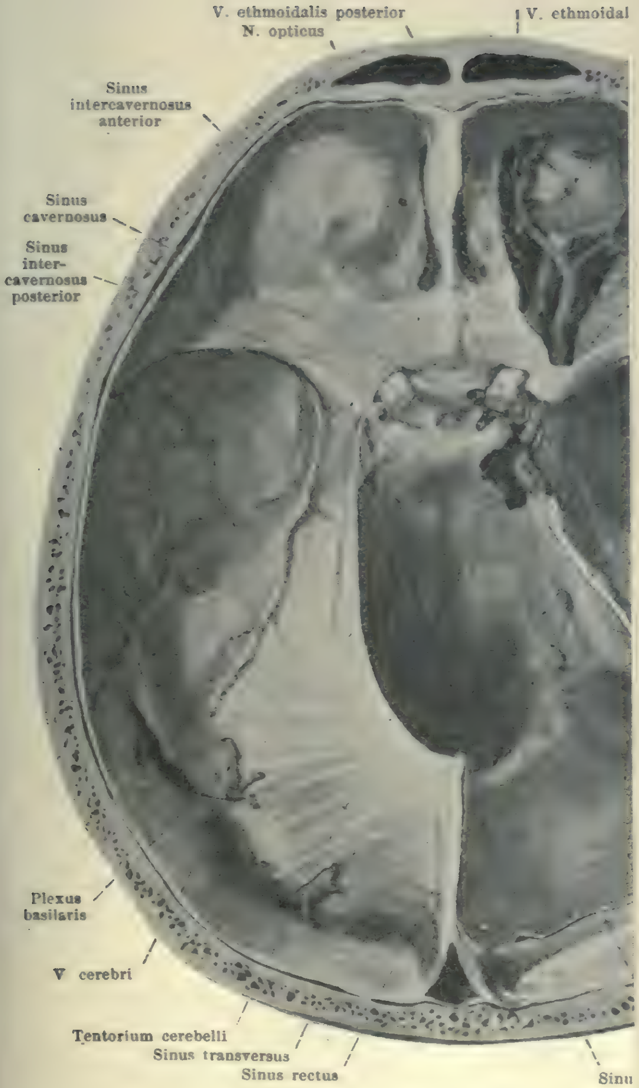

V. ethmoidalis posterior
N. opticus
V. ethmoidal
Sinus intercavernosus anterior
Sinus cavernosus
Sinus intercavernosus posterior
Plexus basilaris
V cerebri
Tentorium cerebelli
Sinus transversus
Sinus rectus
Sinu

FIGURE IX.

XXI.

RESULTS IN A SERIES OF CASES OF TONSILLECTOMY AT THE MASSACHUSETTS GENERAL HOSPITAL, THREE TO FOUR YEARS AFTER OPERATION.*

By J. Payson Clark, M. D.,

Boston.

The year beginning July 1, 1908, was chosen for study because sufficient time has elapsed since then to make the reports valuable as end results. The reasons for not selecting an earlier year were, in the first place, that this was about the time when we began to do tonsillectomy at the hospital as a practically routine procedure in all cases where a removal of the tonsils appeared to be indicated; and in the second place, because the longer the time since the operation the smaller the number who were likely to respond to a request to report. The reasons for this latter statement are obvious and need not be mentioned. Postal cards stating, in substance, that Dr. Clark was interested to know their present condition and would like to have them report on one of certain given dates at the throat clinic of the hospital, were sent to all the cases. Those selected were under the heading "adenoids and tonsils" in our card index. Of course many of these cases had only an adenoid operation, and such cases were not asked to report. As a result of selecting only the cases of tonsil operation which were listed under this heading, it is obvious that adults were practically excluded from the list. It was felt that as the tonsil is physiologically more active in children, a study of end results would be more valuable in them, and that the results of tonsillectomy in adults could be more profitably considered in a separate paper.

I began sending out these notices in July, 1912, sending out a few each week, so that I should not be overwhelmed by too large a number reporting at any one time. I wanted to see all the cases myself, but this was not possible, as I was unable to go to the hospital at several different times. The cases which I did not see personally (fifty-three in all) were examined by one of my colleagues and recorded for me.

---

*Read at the meeting of the American Laryngological Association in Washington, May, 1913.

Nine hundred and ninety-two postals were mailed, and to these one hundred and forty-three patients responded by reporting in person at the clinic. This is only 14.4 per cent of the number asked to report. This percentage is a little better than a few years ago, when I wrote on "Results in Adenoid and Tonsil Operations" (*Boston Medical and Surgical Journal,* September 24, 1908). At that time I sent out five hundred and ninety-five cards, and (to quote from that paper) "the responses were exceedingly disappointing as to number, amounting in all to seventy-three, or a little over 12 per cent of the cases to whom notices were sent. Between three and four years having elapsed since the operation, many of the patients have doubtless moved, so that the cards did not reach them, and others live at too great a distance to be able to come in easily." In talking to other investigators of all kinds of medical and surgical cases, I find that my experience in this particular is a common one. I believe, however, that it is fair to take the cases which did report as a pretty good criterion of the results in all the cases. I received a dozen or more letters in response to the card, stating that the patient could not come to the hospital for one reason or another. These cases are, of course, not included in my statistics. Those who reported were subjected to the following examination:

1. Patient's name, sex, age (at present time), number?
2. Date of operation?
3. Any hemorrhage after operation requiring treatment? If so, what treatment?
4. Was adenoid removed at same time or not?
5. For what reason were the tonsils removed? (On account of size, frequent tonsillitis, enlarged cervical glands, rheumatism or other evidence of septic absorption or any other reason?)
6. Was the trouble for which the tonsils were removed relieved?

7. Was there a marked improvement in general condition after operation?
8. Has the patient had any illness since operation? If so, what and when? Has patient had any sore throats?
9. Present state of health?
10. Appearance of throat? (State whether any tonsillar tissue appears to remain, also as to symmetry of soft palate and pillars, presence or absence of tonsillar fossæ, etc.)
11. Any difference in voice or speech since operation?
12. Are there any enlarged cervical glands now?

A review and analysis of the answers to the questions and the findings at the examination follow:

The ages of the patients ranged from five to thirty-six years, distributed thus: 5 to 10 years, 45; 10 to 15 years, 76; 15 to 20 years, 17; over 20 years, 4. As these are the ages at the present time, it is obvious that the ages averaged four years less at the time of the operation. Of the patients over 20 years of age, one is now 23, one 26, one 27 and one 36 years old. There were seventy-six males and sixty-seven females. All who reported had adenoids removed at the same time. It is hardly necessary to say that the adenoids were of varying relative importance in the different cases, sometimes being so small as to seem to be hardly worth considering, and in other cases being the chief reason for operation.

Only one case had bleeding after the operation which was worthy of note. In this case the bleeding was so free that the patient was kept in the hospital over night, but no operative treatment was required to stop the bleeding. I might say here that up to last fall it was our custom at the Massachusetts Hospital to allow children on whom tonsillectomy had been done to go home in the afternoon (the operation having been done in the morning). It was felt that if there was no evidence of bleeding by that time, it was fairly safe to asume that there would not be any. On account of the small number of beds at our disposal, we were forced to take this risk or else turn away the majority of our tonsil cases in children. An exception, however, was made in the event of children living at such a distance from the hospital that they could not be easily reached in case of secondary hemorrhage. These statistics tend to show that the risk which we took was a small one. Since last fall we have had more beds put at our disposal, and now keep all our tonsillectomy cases in the ward for one night. This routine is certainly more conducive to one's peace of mind and much fairer to the children. For, although I feel that where tonsillectomy is competently and carefully done, hemorrhage coming on after the patient has left the operating room and requiring treatment is really a rare occurrence, yet when it does occur it is often so alarming and prompt attention is so important that to keep such patients under observation for twenty-four hours is not being overcautions.

The following table shows for what reason the tonsils were removed and whether the conditions were relieved or not:

Unknown	15
Sore throat relieved	10
Sore throat not relieved	4
Sore throat cured	29
Size	96
Adenitis cured	15
Adenitis not cured	17
Tonsillitis cured	32
Tonsillitis not cured	1
Chorea improved	1
Rheumatism cured	2
Speech cured	1
Circumtonsillar abscess cured	3

Of the fifteen cases in which the reason for removal is not known, several were no doubt operated on because they were under an anesthetic for an adenoid operation, and it was thought just as well to take the tonsils out, without considering whether there were good reasons for their removal or not. We endeavor to guard against unnecessary removal of the tonsils by requiring that the man who operates shall see that the reasons for removing the tonsils are distinctly stated on the history.

There was a marked improvement in the general condition after the operation in eighty-nine cases, a moderate improvement in twenty-nine cases and no improvement in twenty-four cases. Since the operation one patient has had nasal diphtheria, four measles, two whooping cough, one neuritis, one "congestion of the lungs," one chorea, one bronchitis, one pleurisy, five abscess of the ears (two of these requiring a mastoid operation), three pneumonia, and twenty-two sore throat. The present state of health is good or excellent in one hundred and eighteen cases and fair in twenty-two cases. There has been no change in the voice or speech in one hundred and sixteen cases. In twenty-two cases the voice or speech has been improved. In two the voice or speech is said to be not so good. Enlarged cervical glands were absent in ninety-eight cases and present in forty-three. In two cases there is no record on this point.

Tonsil tissue was absent in eighty-two cases. Some tonsil tissue was still present on both sides in thirty-one cases, and on one side only in twenty-eight. Examination was unsatisfactory in the case of an unruly child. Certain cases where it was doubtful whether or not the appearance in the tonsillar fossæ was due to tonsillar tissue, were listed as showing it. In some of these cases it is fair to assume that the lymphoid tissue which was apparently present in the fossa was due to the hypertrophy of some focus of lymphoid tissue which lay outside the tonsil capsule.

The soft palate was symmetrical in one hundred and twenty cases and asymmetrical in eighteen. In five cases no note was made on this point. The soft palate was considered asymmetrical when the uvula was at all to one side of the median line, or when one arch of the velum was flatter than the other. The pillars of the fauces were normal in ninety-six cases and not normal in forty-one. There was no note on this point in six cases. The pillars were recorded as not normal when one or more of them were absent, or when the anterior and posterior pillars on the same side were fused, or when scar tissue showed that one or more of them had been injured at the operation. The tonsil fossæ were present on both sides in ninety-five cases, shallow in sixteen, and one or both were absent in twenty-six cases. There was no record of their condition in five cases. Carious teeth were noted as present in twenty-one cases. Three cases which had enuresis before the operation were not relieved of this trouble. In four cases the uvula was partly or wholly cut off.

Now, let us analyze some of these results and see if we can obtain any interesting or valuable facts from them. In the fifteen cases in which the reason for removing the tonsils does not appear, they may have been removed because they were visible and the operator was of the opinion that as the patient was under ether for an adenoid operation the tonsils were better out, too. Or it may have been that there was some excellent reason for their removal which the operator failed to record. I have already stated how we try to guard against any unnecessary removal of the tonsils.

"Sore throat" was given as a reason for removal of the tonsils in cases in which it was not clear as to whether there had been definite tonsillitis, yet on inspection it appeared probable the tonsils were the seat of the trouble. These numbered forty-three in all. Twenty-nine of these cases have had no sore throat since the operation, ten have had it less frequently or less severely, and four say they have been no better. Of the ten cases which were relieved, one, a woman of 27 years of age, had suffered from frequent tonsillitis before, but since the operation says she has slight sore throat at times. An examination of this case showed in the fossæ a very small amount of tissue with a suggestion of lymphoid characteristics. The patient had not reported at the time of these attacks of sore throat, and it seems fair to assume that they might have been simple pharyngitis. Of the remainder, five showed the presence of more or less tonsil tissue in one or both fossæ. But in all these cases the sore throat was so slight and so infrequent as not to require medical attendance or keep the patients from their regular employment. Four, who have occasional slight sore throat, showed no tonsil tissue. The sore throat in these cases was probably a pharyngitis. Of the four cases in which the sore throat was said to be no better, two showed absolutely no remains of either tonsil, and one of these had a marked chronic pharyngitis. One of the other two showed a small remnant of tonsil on the right, but it seemed very doubtful if this could be the origin of the sore throats which the patient said he had, but for which he had never applied for treatment. In the remaining case there was some doubtful lymphoid tissue in the right fossa.

Of the thirty-two cases in which cervical adenitis was mentioned among the reasons for tonsillectomy, fifteen were cured, i. e., they show no enlarged cervical glands now. A further consideration of the seventeen cases of adenitis which are put down as not cured, discloses some interesting facts, But it will perhaps be more satisfactory to consider these cases later in connection with those in which the presence or absence of cervical glands was not mentioned at the time of the operation, but in which cervical glands were felt at this examination.

Of the thirty-three cases in which frequent tonsillitis was given as a reason for tonsillectomy, only one was not cured, and in this case it was found that the tonsils had been only partially removed. A case which came on account of deafness was cured, but the simultaneous removal of the adenoids was no doubt a large factor in this result. One case of chorea is much better, but not completely cured. Two cases of rheumatism are cured. It is only fair to say that in neither of these cases was the history of rheumatism very clear. Three cases of circumtonsillar abscess have been cured, one being now 36 years old, one 26, and one 17. Only one patient under 21 years old had circumtonsillar abscess. In eighty-nine cases there was marked improvement in the general condition after the operation. As the adenoids were removed at the same time, a part of the improvement must be attributed to this cause in many of these cases. In twenty-nine other cases there was some improvement, but not marked. In twenty-four cases there was no improvement in the general condition. In fourteen of these cases the health was good before the operation, and the reason for operation was a purely local one. One case of quinsy is cured of that affection and has no tonsil tissue remaining. She now complains of pain going to different parts of the body, on which no diagnosis has been made. One case has no tonsils, but a marked nasal obstruction due to a deviated septum, which may account for lack of general improvement. One case shows no tonsils, but very bad teeth, which are undoubtedly the cause of poor general condition. One patient, whose tonsils have been completely removed, is pale and nervous from some other cause, probably improper diet and carious teeth. One case of retarded mental development was not improved by the operation. One patient, described as "nervous and fidgety" by her mother before the operation, shows no improvement in this respect. Her tonsils have been completely removed. The neurologist describes the condition as an "involuntary twitching of the shoulders, characteristically repeated: diagnosis, habit spasm." Four of these cases still show presence of some tonsil tissue on one or both sides. In one of the four it seems more than probable that the present anemic condition and loss of appetite is due to bad teeth and chronic rhinitis, rather than to the small amount of the lower lobe of each tonsil which appears to be present. In the other three cases it is fair to assume, in the absence of other obvious cause, that the lack of general improvement is due to the incomplete operation.

Among the cases which reported there have been the following illnesses: nasal diphtheria, one; measles, four; whooping cough, two; pneumonia, three; "congestion of the lungs," one; pleurisy, one; bronchitis, one; neuritis (?), one; chorea, one; abscess of the ear, five cases (two of which had a mastoid operation). Several had more than one illness, so that the actual number of individuals in the above list is only sixteen. It strikes me as a remarkably good record that of one hundred and forty-three children only sixteen have had a definite illness in four years. The present state of health is stated as good or excellent in one hundred and eighteen cases, fair or poor in twenty-two. Ten of these latter showed definite remains of tonsil in one or both fossæ, while three were doubtful. The presence of some tonsil tissue may account for the poor health in some of these thirteen cases, while in the nine others in which there is no tonsil remaining, one has chorea; three, bad teeth; two, nasal obstruction; and one has just recovered from an illness.

There was no change in the voice or speech in one hundred and sixteen cases, including the four in which the uvula had been partially or wholly cut off. This accident has had no ill effect, save a cosmetic one, in any case. In twenty-two cases the voice or speech was improved, i. e., it is clearer or more distinct or of better quality. In two, the voice or speech is said not to be so good. One boy talks somewhat through his nose, which he is said not to have done before the operation. His soft palate, uvula and faucial pillars are normal, but he has a nasal obstruction which probably accounts for the nasal voice. The other is said to talk at times through the nose, but there is no apparent reason for this, as his throat is perfectly normal.

Forty-three cases showed one or more enlarged cervical glands. In not one of these cases were the glands causing any apparent symptoms or inconvenience of any sort. They were discovered on palpation of the neck. In seventeen cases a few small glands were present on both sides. In thirteen of these tonsil tissue was left on both sides. In three of the thirteen there were also decayed teeth. In five there were decayed teeth and no tonsil. In five others there was no tonsil tissue and no decayed teeth. In four cases there were several glands, and in eleven cases only one palpable gland under the left angle of the jaw. Eight of these fifteen cases showed the presence of a remnant of the tonsil on the left, and in one there was also a decayed tooth. In four there were decayed teeth and no tonsil, and in two there were no tonsil and no decayed teeth.

In three cases there were several and in five cases one gland on the right. In three of these eight cases there was some tonsil remaining on the right, and in three no tonsil, but decayed teeth. In these cases it is interesting to note that in most of the cases where there were glands present, there was still some tonsil on the same side. In five of the cases in which glands were felt, it was doubtful whether or not tonsil tissue remained. On the other hand, in twenty-six cases in which there were remains of the tonsil on one or both sides, there were no enlarged cervical glands. In four of the cases in which there were decayed teeth there were no enlarged glands.

Case.—S. B. F., female, age 8½ years, was seen in the neurologic department in March, 1909, where the following note was made: "Patient has general chorea, twitching of both arms and both legs so that she cannot walk, her mother says, or feed herself. Heart negative. Advise operation for tonsils." She also gave a history of frequent tonsillitis. Her tonsils were removed on April 1, 1909. When she came in to report it was stated that she was much improved in choreaic symptoms, but not cured. On examination, no tonsil tissue could be seen. She had one or two teeth with cavities and one pea-sized gland under the left angle of the jaw. She was sent to the dentist.

SUMMARY.

The patients, with few exceptions, were under fifteen years old at the time of operation.

Hemorrhage after tonsillectomy, calling for special treatment, is of rare occurrence.

The condition for which the tonsils were removed was relieved in very nearly every case, even in those in which there is still some tonsil tissue.

An improvement in the general health is to be expected after tonsillectomy done for cause.

Children who have had tonsillectomy certainly show no increased tendency to illness, and are probably less susceptible than before operation.

Present health is excellent in the great majority of cases.

What is apparently tonsil tissue is found much more often than one would expect after supposed tonsillectomy.

The soft palate was symmetrical and the faucial pillars and tonsil fossæ normal in the great majority of the cases.

The accidental cutting off of the uvula in four cases caused no bad symptoms.

Most of the cases of tonsillitis and sore throat were relieved by the operation.

In many of the cases in which there appeared to be remains of tonsil tissue the patients are in perfect health, and in others in which there were symptoms, they were undoubtedly due in many instances to other causes than the tonsil remnants.

The ordinary voice or speech may be said to be practically unaffected by tonsillectomy. No investigation was made as to the singing voice. In most of the cases in which enlarged cervical glands could be felt, there was tonsil tissue present on the same side.

On the other hand, in nearly half the cases in which tonsil tissue is still present, there are no enlarged glands.

Carious teeth were apparently a direct cause of some cases of cervical adenitis.

XXII.

REPORT OF REMOVAL OF FIBROMYOMA WHICH EXCLUDED THE AUDITORY CANAL; HEMOPHILIA.*

By M. D. Lederman, M. D.,

New York.

Mrs. N., 30 years of age, had had chronic suppurative otitis since early childhood. Ten or more years ago I removed a large growth from the left ear, and at that time there was considerable difficulty in arresting the bleeding. The ears ceased discharging after the operation for some years, and I did not see the patient again until three months ago, when she consulted me on account of a purulent bloody secretion from the same ear. She remarked that the ear had been moist for some time, but that she kept it quite clean by douching and a cotton applicator, but that of late she noticed that she was not able to get into the canal, as something obstructed the passage of the applicator. Furthermore, she had occasional headaches, and recalling her former trouble she sought my advice.

Examination showed the canal to be filled with the growth, the free end projecting from the opening of the meatus. Some foul-smelling secretion was found in the canal. No inflammatory symptoms of neighboring tissues were present. Prompt removal of the fibrous polyp was advised, to avoid retention of accumulating secretion.

As the patient desired a few days to get her household affairs in readiness, knowing that she would be detained in the hospital in case the bleeding should be unusual in character, calcium lactate was prescribed for four days, to increase the coagulability of the blood. She informed me that on one occasion a tooth had been extracted and that the dentist had experienced considerable difficulty in arresting the hemorrhage. Furthermore, she suffered from prolonged and excessive menstrual periods. She also stated that when she struck herself against any hard substance on different parts of her body, subdermal ecchymosis resulted.

---

*Read before the New York Academy of Medicine, Section on Otology, February 14, 1913.

The technic of removing the large polyp was simple. Tampons soaked with cocain were placed around the attachment of the growth, as near the middle ear as they could be carried, and a few drops of a half per cent cocain solution were injected in the upper and inner portion of the meatus. The snare was readily carried around the growth to the membrana, and then drawing the wire taut a quick jerk of the instrument brought away the growth, which was over an inch long and a quarter of an inch in thickness. There was but little bleeding immediately following the removal of the growth, and I was about to laud the virtues of calcium lactate as a clot promoter. In a few minutes, however, a little blood began to ooze from the canal, so a packing of adrenalin-soaked gauze was introduced. This had no effect, as the blood continued oozing through the gauze without clotting. Other remedies were tried without success until 10 cc. of Mulford's horse serum were injected into the arm, and although the needle puncture bled for a few minutes, the oozing from the ear ceased in twenty minutes.

The canal was repacked, for safety, and the patient was transported to the Polyclinic Hospital for observation.

On attempting to clean the ear the following day, the bleeding again started, but was controlled by repacking. Within forty-eight hours, large urticaria of a hemorrhagic type were observed on the patient's right thigh and at the point of injection. These remained for over a week, causing considerable annoyance by the intense itching. This condition was no doubt due to the serum (anaphylaxis).

The ear soon became dry, and hearing was much improved. We must not overlook the fact that in the removal of these large fibroid polyps from the middle ear we might stimulate an existing infection in this cavity which may set up very serious complications. Mastoiditis and meningitis have followed the extraction of these hypertrophic tissues.

XXIII.

ABSCESS OF FRONTAL LOBE OF THE BRAIN, OF OTITIC ORIGIN, WITH EXHIBITION OF SPECIMENS.

By T. Passmore Berens, M. D.,

New York.

If the case of abscess in the frontal lobe of the brain that I am about to present to you is one of otitic origin, then—so far as I have been able to learn from a comprehensive search of the literature reaching back to 1869, made by Dr. A. Braun—it would seem to be unique. He has gathered together forty-nine cases, excluding the one that I present, of abscess in the frontal lobe, none of which was of otitic origin. Case No. 28, while presenting symptoms of mastoiditis, may be classified as a case of direct infection by extension from the ethmoidal labyrinth. I have given a short extract of each case, with its reference; but in order to save the time of this society I have also prepared a short table of the cases.

1. Frontal sinusitis.—Early headache; apathy; rigidity of neck. Autopsy: Abscess frontal lobe.

2. Frontal and ethmoidal sinusitis.—Persistent headache; optic neuritis; pulse 55 to 60; somnolence. Recovered after operation—frontal lobe abscess.

3. Frontal sinusitis.—Recovered after operation—frontal lobe abscess.

4. Frontal sinusitis.—Early headache: pain: temperature 100½°; increasing vomiting; incontinence urine: constipation. Autopsy: Abscess frontal lobe.

5. Pulmonary tuberculosis.—Conjugate deviation; pupils dilated; temperature 100 1/5°; delirium: stupor: hemiplegia. Autopsy: Multiple abscesses in frontal lobe.

6. Fractured base.—Pain in eye: apathy: hemiplegia: vomiting. Autopsy: Abscess frontal lobe.

7. Unknown etiology.—Headache; malaise; chills; temperature 99½°. Autopsy: Abscess frontal lobe.

8. Fractured skull.—Spasms of face extending to arm and leg; temperature 103.° Autopsy: Abscess frontal lobe.

9. Healed gunshot wound of head.—Headache; pupils unequal; stupor; twitching and slight hemiplegia; dysphagia. Autopsy: Abscess frontal lobe.

10. Frontal sinusitis.—Headache and vomiting; temperature 37½° C.; venous hyperemia of eyes. Operation revealed abscesses frontal lobe. No autopsy.

11. Ethmoiditis.—Apathy; exophthalmos; delirium. Autopsy: Abscess frontal lobe. Basilar meningitis.

12. Ethmoiditis.—Headache; vertigo; vomiting; apathy; hemiplegia. Autopsy: Abscesses frontal lobe. Hemisphere adherent to lamina cribrosa.

13. Nasal.—Abscess frontal lobe.

14. Fracture frontal bone.—Apathy; incontinence of urine; prolapse of brain tissue—removed by thermocautery opening anterior horn of lateral ventricle; three and one-half months later bulging of scar; erotic mania and convulsions; spontaneous rupture. Disappearance of symptoms.

15. Frontal sinusitis.—Abscess frontal lobe.

16. Frontal sinusitis.—Multiple abscesses frontal lobe.

17. Panophthalmitis.—Temperature 39° C.; coma. Autopsy: Diffuse meningitis. Abscess frontal lobe.

18. Frontal sinusitis.—Optic neuritis; coma; hemiplegia; headache. Recovery after three operations for recurring abscesses in frontal lobe.

19. Trauma.—Headache; apathy; emaciation; hemiparesis; neuritis; pulse and temperature normal; symptoms of meningitis. Operation: Abscess frontal lobe. Recovery. One year later recurrence; unconsciousness; death. No autopsy.

20. Trauma of orbit.—Convergent squint; apathy; loss of memory; later proptosis; choked disc; temperature subnormal; pulse 80. Operation: Abscess frontal lobe. Recovery. Later, operation motor area abscess. Recovery.

21. Tubercular bronchiectatic cavities.—Facial paralysis; Jacksonian epilepsy, beginning right arm; hemiplegia. Autopsy: Two abscesses frontal lobe.

22. Tubercular bronchiectatic cavities.—Mental dullness; apathy; temperature 97 to 99°; pulse 84; no other symptoms of brain involvement. Autopsy: Abscess frontal lobe.

23. Frontal sinusitis.—Irritability; optic neuritis. Operation: Abscess frontal lobe. Recovery.

24. Frontal sinusitis.—Slow cerebration; pulse 48. Operation: Abscess frontal lobe. Recovery.

25. Pansinusitis.—Operation: Epidural abscess. Frontal lobe abscess. Death.

26. Ethmoiditis.—Paralysis arm and leg; coma. Operation: Frontal lobe abscess. Death.

27. Frontal sinusitis.—Intermittent headaches; convulsions. Two operations. Frontal abscess. Recovery.

28. Ozena. Acute Mastoid.—Symptoms: Mental dullness; headache. No operation. Autopsy: Abscess frontal lobe with dura adherent to ethmoid.

29. Frontal sinusitis.—Headache; apathy; temperature subnormal; pulse 56. Operation. Autopsy: Frontal lobe abscess.

30. Frontal sinusitis.—Dullness of intellect. Operation: Frontal lobe abscess. Unconsciousness; hemiplegia. Second operation: Motor area. Abscess found. Death.

31. Traumatic ethmoiditis.—No localizing symptoms. Autopsy: Necrosis cribriform plate. Frontal lobe softened and infiltrated with pus.

32. Pansinusitis.—Headache; delirium. Operation: Cyst of frontal lobe. Autopsy: Basilar meningitis. Abscess frontal lobe.

33. Frontal sinusitis.—Stupor; vomiting. Operation: Frontal lobe abscess. Recovery.

34. Gunshot wound.—Headache; irritability; optic neuritis left. Retrobulbar neuritis right; increasing hemiplegia. Operation: Frontal lobe abscess. Recovery.

35. Traumatism.—Temperature 99 to 104°; arm and leg paralyzed; retinal vessels tortuous; hemiplegia; coma; convulsions. Operation: Frontal lobe abscess.

36. Frontal sinusitis.—Headache; one-sided convulsions. Operation: Abscess frontal lobe. Death.

37. Perforating trauma.—Vomiting; general convulsions. Autopsy: Abscess frontal lobe.

38. Trauma.—Baby ten and one-half months old. Severe pain; twitching and paralysis arm and leg; fever; coma; pupils equally dilated; circumference of head increased. Autopsy: Abscess frontal lobe.

39. Trauma.—Vomiting; coma; tremor one side. Autopsy: Abscess frontal lobe.

40. Trauma.—Twitching arm and leg; epileptiform seizures. Operation: Abscess frontal lobe. Recovery.

41. Penetrating trauma.—Temperature 39° C.; pulse 45; right hemiplegia; left ptosis. Operation: Abscess frontal lobe. Recovery.

42. Necrosis frontal bone.—Vomiting; fever; convulsions; anesthesia of left face; diminished hearing left; paralysis left sixth nerve; loss of speech; ulceration of cornea; coma. Autopsy: Extensive necrosis of frontal bone; purulent meningitis. Abscess frontal lobe.

43. Frontal sinusitis.—Headache; convulsions; hemiparesis. Operation: Brain hernia formed, then removed, liberating pus. Temporary relief (one month). Convulsions six months. Coma. Death. Autopsy: Frontal lobe abscess. Purulent basilar meningitis.

44. Trauma of orbit.—Convulsions. Operation: Frontal lobe abscess. Recovery.

45. Cause unknown.—No paralysis; no convulsions. Clinical picture of tubercular meningitis. Frontal lobe abscess.

46. Trauma.—Headache; somnolence; vomiting; pulse slow; respirations slow; temperature normal; patellar reflexes diminished; pupils unequal; paresis left internal rectus; double optic neuritis; convulsions; pleurosthotonus. Operation: Abscess frontal lobe. Recovery.

47. Trauma.—Headache; somnolence; temperature 100°; pulse 60; vomiting; Jacksonian epilepsy; hemiplegia; temporary aphasia. Autopsy: Abscess frontal lobe.

48. Acute ethmoiditis.—Mentality dull; knee-jerk absent; optic neuritis. Autopsy: Pus in ethmoid. Abscess frontal lobe.

49. Orbital abscess.—Headache; vomiting; pulse 64; temperature normal; Jacksonian epilepsy. Autopsy: Frontal lobe abscess and orbital abscess.

Case 1.—Redtenbacher: "Hirnabszesse und Eiterung im Sinus frontalis nach Influenza." *Wiener medizinische Blätter,* 1892, 13. Female, 34 years of age. Had an attack of influenza. A few days later redness and swelling of left upper eyelid. Then clonic spasms. Headache. Three weeks later, weakness of right half of body. Abscess of eyelid was opened. Did not heal. Apathy and rigidity of neck. Death three months later. Postmortem showed pus in the frontal sinus, with necrosis of its posterior wall. Dura adherent to this wall. At the apex of the frontal lobe, a nut-sized abscess.

Case 2.—A. Denker: "Rhinogener Frontallappenabszess und extraduraler Abszess in der Stirngegend, durch Operation geheilt." *Archiv. f. Laryng. u. Rhin.*, 1900. Male, 17 years of age. Pain in region of left temple and eye. Foul odor from nose. Diagnosis of left frontal sinusitis and ethmoiditis was made. Intranasal operation. A month later, external operation. Two weeks later the headaches became worse, there was somnolence, there was blurring of the left optic disc, and the pulse was 55 to 60. The posterior wall of the frontal sinus was removed, and an epidural abscess was exposed. The dura was incised. Brain tissue was aspirated and pus found. Brain incised and abscess evacuated. Recovery.

Case 3.—J. Herzfeld: "Rhinogener Stirnlappenabszess, durch Operation geheilt." *Berliner klinische Wochenschr.*, 1901, No. 47. Male, 20 years of age. Six weeks before admission he had coryza. Two weeks ago, left-sided frontal headaches. Diagnosis of left frontal sinusitis was made. External operation. Posterior wall of frontal sinus was found carious and removed. An epidural abscess was found. The dura was incised. A subdural abscess was evacuated. When this pus was wiped away, a fistula was seen in the frontal lobe of the brain. The fistula was enlarged with forceps, and a finger introduced into the abscess cavity. Iodoform gauze packing. Recovery.

Case 4.—E. S. Steese: Medical and Surgical Report of the Presbyterian Hospital, 1898. (1)—C. S., 17 years of age. Six weeks ago sudden severe pain in both eyes, dimness of vision and vomiting. Pain lasted three weeks and then vanished. Mental dullness, delirium and stupor. Increasing difficulty in swallowing and breathing. Patellar reflexes absent. Pupils dilated. Pulse about 50, temperature 100.5° F. Vomiting, incontinence of urine, constipation, death. Postmortem showed frontal sinus disease. Abscess in left frontal lobe, three-fourths inch in diameter. There was a perforation in posterior wall of frontal sinus.

Case 5.—(2) C. S., 39 years of age. Phthisis for several months. Two weeks ago. paralytic stroke, during sleep. Delirium and stupor. Left hemiplegia. Occasional clonic spasm of left side, with spasmodic conjugate deviation. Right patellar reflex increased, and ankle clonus. Pupils dilated. Respiration stertorous. Temperature averaged 100.2° F. Death four weeks later. Postmortem showed an abscess one inch in diameter at upper end of fissure of Rolando. Another in occipital lobe. A large cavity in left frontal lobe.

Case 6.—(3) A. H. Fracture at base of skull. Trephined in frontal region, and considerable fluid withdrawn. Six months later pain over right eye. Apathy and left hemiplegia. Patellar reflexes lost. Respirations shallow. Vomiting. Death. Postmortem disclosed a large abscess in right frontal lobe. A few foci of suppuration were also found at the top of the left parietal lobe.

Case 7.—(4) B. J., 25 years of age. One week ago headache, malaise and chills. Temperature averaged 99.5° F. Death after a week. Postmortem showed flattening of the convolutions of the brain, and an abscess one inch in diameter in left frontal lobe. Entire lobe softened. Etiology unknown.

Case 8.—(5) Female. Was struck by cable car and received a linear fracture of the skull. Spasms, commencing in left side of face and extending to arm and leg. No paralysis. Temperature averaged 103° F. Duration three days. Death. Postmortem disclosed two large abscesses in the frontal lobe. Laceration of cerebellum. Pneumonia. Mucopus in smaller bronchi of both lungs.

Case 9.—(6) M. S., 45 years of age. Bullet removed from head ten months ago. Headache, stupor, twitching of right hand and leg, and slight paralysis of left side. Difficulty in swallowing. Right pupil dilated, and left contracted. Rigor. Death. Postmortem: Entire right frontal lobe honeycombed with abscess cavities.

Case 10.—M. Paunz: "Ueber den rhinogenen Hirnabscess." *Arch. f. Lar. u. Rhin.*, 1902-3, Vol. 13. Female, 16 years of age. Acute left frontal sinusitis. Headache and vomiting. Temperature 37.5° C. Pulse 60 to 66. Dilated, sluggish pupils. Venous hyperemia of the eye grounds. Operation: Small frontal sinus was found. Dura was exposed and found bulging. Aspirated pus through dura. Incision of dura and brain substance. About 80 cc. of pus evacuated. Death six months later.

Case 11.—H. Maas: "Zur Casuistik und Therapie der Gehirnabszesse nach eigenen Erfahrungen." *Berliner klin. Wochenschr.*, 1869. Male, 45 years old. Nasal polypi. Removed several times. Frequent nosebleed. Apathy. Vertigo. Nasopharyngeal polyp. This, as well as the nasal polypi, removed. Patient was somewhat relieved. Followed after several days by increase in the apathy. Exophthalmos, delirium and death. Postmortem showed dura at the base of the skull covered with pus. The lamina cribrosa and upper surface of the sphenoid was destroyed. Abscess in the frontal lobe of the brain, above ethmoidal region.

Case 12.—D. Jacubasch: "Abszess im vorderen rechten Gehirnlappen." *Berliner klin. Wochenschrift*, 1875. Male, 20 years old. Had sore in nose. Dull headache, vertigo and vomiting. Speech slow and difficult. Pulse 56. Evening temperature 38° C. For several weeks no change. Then apathy. Pain in back of neck. Left-sided hemiparesis. A month later, death. Postmortem: Right hemisphere adherent to lamina cribrosa. Large abscess in right frontal lobe. A second abscess further back.

Case 13.—Seifert: "Abscess of frontal lobe of nasal origin. *Revue hebd. de laryngol.*, etc., 1905.

Case 14.—Borchard: *Arch. f. klin. Chirurgie,* 1904, Vol. LXXIV. Female, 8 years old. Was kicked in left side of forehead by horse's hoof. Frontal bone fractured. Brain tissue exposed. Loose fragments of bone were removed. Dressing. Child apathetic. Passed urine involuntarily. Prolapse of brain tissue removed with thermocautery. This opens up anterior horn of lateral ventricle. Patient discharged six months later. Three and a half months later she was readmitted. Five days before her readmission the old scar began to bulge. She developed symptoms of erotic mania, and had several convulsions. The swelling ruptured spontaneously and evacuated pus. This was followed by disappearance of the mental symptoms. The swelling with recurrence of the mental symptoms reappeared several times in the course of the next few years.

Case 15.—Cisneros: "Abscess of the frontal lobe following frontal sinusitis." *Revue hebd. de laryngol.*, etc., 1905.

Case 16.—R. Hoffmann: "Multiple abscesses of the brain, resulting from bilateral traumatic frontal sinusitis." *Arch. internat. de laryngol.*, 1906.

Case 17.—W. Reis: "A case of panophthalmitis with brain abscess and fatal meningitis." *Arch. f. Augenheilk.*, 1905. Male, 37 years of age. Was struck in left eye with stick a week before. Panophthalmitis. Eyeball was incised and pus evacuated. Five days later, severe headache, temperature 39° C., coma, and death the same evening. Postmortem showed a diffuse meningitis and an abscess in the anterior part of the frontal lobe.

Case 18.—L. B. Rawling: "Case of Chronic Abscess of the Frontal Lobe." Transactions of the Medical Society of London, 1907. Male. Frontal headache with edema of the right upper eyelid, in May, 1905. Roof of orbit was exposed and found bare of periosteum. On June 1st, coma and paralysis of left side of body and face, vomiting, optic neuritis, slow pulse and subnormal temperature. Skull was opened so as to expose outer and anterior aspect of frontal lobe, and an abscess was found. Rapid recovery. Discharged cured in the middle of July. In 1906, return of the same symptoms. A similar operation was performed and another abscess found. Recovery. On October 13, 1906, he was admitted for the third time. Marked bulging at the site of former operations. Drowsy and irritable. Severe headache. Left hemiplegia. Abscess was not found. Marked hernia. On October 24, again operated and abscess found. Recovery. At a later date frontal sinus was opened and found to contain pus and carious bone.

Case 19.—F. L. Taylor: "A Case of Abscess of the Frontal Lobe of Traumatic Origin." *New York Medical Journal,* May, 1908. Male, 27 years of age. Was struck with a brick above the right eye in May, 1906. Was not rendered unconscious. A month later, superficial abscess was opened. Later, headache, apathy, emaciation, optic neuritis, slight paresis of the left side of the face, and weakness of grip in left hand. Stiffness of back of neck, vomiting, incontinence of urine and feces. Pulse and temperature normal. Operation showed abscess in frontal lobe on right side. Recovery, with poor vision. In October of the following year he became unconscious, had several convulsions, and died.

Case 20.—Coulter: *Ophthalmoscope,* 1909. Female, 24 years of age. Piece of wood, one and one-half inches long, was driven into the left orbit, through the upper eyelid. Removed. This was followed by a convergent quint and diplopia. Swelling in upper outer angle of orbit. Incised. Three months later: apathy, loss of memory, pulse 80, temperature subnormal. Both eyes were proptosed. Left external rectus was paralyzed. Choked disc. The skull was trephined behind the external angle of the left orbit. Abscess was found in frontal lobe. A month later, right arm and leg began to twitch. Trephined over motor area, and another abscess tapped. Cure.

Case 21.—Schorstein: *Lancet,* 1909. (1) Male, 33 years of age. Tuberculosis, with bronchiectatic cavities. Coma. Facial paralysis left. Muscular spasm, beginning in right arm. Seven days later, hemiplegia of right side. Three days later, death. Two abscesses were found in left frontal lobe on autopsy.

Case 22.—(2) Male, 21 years of age. Tuberculosis, with bronchiectatic cavity. Dullness and apathy. No other symptoms of brain involvement. Pulse 84. Temperature 97° to 99° F. Postmortem showed abscess in right frontal lobe.

Case 23.—L. V. Cargill—Wm. Turner—St. Clair Thomson: "A Case of Abscess in Left Frontal Lobe Originating from Suppuration in Left Frontal Sinus." Proceedings of the Royal Society of Medicine, 1907-1908. Male, 31 years of age. Patient had an orbital cellulitis, which was incised. Fourteen days later, irritability and optic neuritis. Frontal sinus was opened and posterior wall found necrotic. Brain covered with granulations. Abscess in frontal lobe evacuated. Recovery.

Case 24.—M. P. Laurens: Proceedings of Parisian Society of Laryngology, Otology and Rhinology. Abstracted in *Journal of Laryngology,* 1908. Slow cerebration, pulse 48. Chronic frontal sinusitis. Operation: Posterior bony wall intact. Infection of brain, either through venous or lymphatic channels. Abscess in frontal lobe, size of mandarin. Recovery.

Case 25.—L. M. Hurd: Transactions of American Laryngological, Rhinological and Otological Society, 1910. Discussion to Dr. McCoy's paper. Chronic pansinusitis, epidural abscess, frontal lobe abscess, meningitis and death.

Case 26.—J. McCoy: Transactions of American Laryngological, Rhinological and Otological Society, 1910. (1) J. L., one and one-half years of age. Ethmoiditis. External operation, Osteomyelitis of vertical plate of frontal bone. Epidural abscess. About three weeks later, paralysis of right arm and leg. Ten days later, coma. Frontal lobe opened. Large quantity of pus evacuated. Death.

Case 27.—(2) H. E., 10 years of age. Frontal sinusitis left. External operation was done by an eye surgeon. Two months later necrotic bone in posterior wall of frontal sinus was removed. Dura covered with granulations. This was followed by intermittent headaches. Two months later, convulsions. Large abscess found in frontal lobe of brain. Five days later, another connecting abscess was found. Counter opening was made in temporal region, and through and through drainage instituted. Recovery.

Case 28.—Thos. Hubbard: Transactions of American Laryngological, Rhinological and Otological Society, 1903. Woman, 25 years of age. Ozena. Acute mastoditis. No operation. Two months later, mental dullness and headache. Mastoid tenderness and death. Autopsy showed dura adherent to under surface of right frontal lobe. Small abscess in center of frontal lobe.

Case 29.—Fagge: Guy's Hospital Reports, 1909, Vol. 63. J. R., 60 years of age. Empyema of frontal sinus. Radical operation. Headache, apathy, subnormal temperature and pulse of 56. Operation showed abscess in frontal lobe on left side. Death. Postmortem showed abscess extending from orbital surface of frontal lobe nearly to anterior horn of lateral ventricle.

Case 30.—J. A. White: Transactions of American Laryngological, Rhinological and Otological Society, 1910. Discussion to Dr. McCoy's paper. Chronic frontal sinusitis. No symptoms of brain involvement, except some dullness of intellect. Operation evacuated frontal lobe abscess. Following operation, unconsciousness. Two days later, lost use of right arm and muscles of face. Opened another abscess in region of Rolandic fissure. Death.

Case 31.—Stucky: ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, June, 1907. Traumatic ethmoiditis, with meningitis and abscess of frontal lobe. No localizing symptoms. Postmortem showed no disease of pia or dura, except over cribriform plate, which was necrotic. Fracture through inner table of left frontal sinus. Anterior and middle cerebral convolutions softened and infiltrated with pale green pus.

Case 32.—Stucky: *Cleveland Medical Journal,* March, 1911. Female, 49 years of age. Pansinusitis. Posterior wall of frontal sinus necrotic. Dura exposed and covered with granulations. Headache and delirium. Improved and was well for three months after operation. Then recurrence of pain. Dura opened and cyst of frontal lobe found, containing one and one-half ounces of thin straw-colored fluid. Died of sepsis four months later. Postmortem: Dura thickened and adherent to brain in frontal and temporosphenoidal region. Base of brain bathed in cloudy fluid. Entire right frontal lobe contained large cavity filled with creamy fluid. Walls of cavity one-fourth inch thick. Anterior half of right temporosphenoidal lobe also disintegrated.

Case 33.—Hans Rische: *Zeitschr. f. Ohrenheilk.,* Vol. 62. Male, 19 years of age. Headache and swelling above left eye. Incised and pus evacuated. Temperature 37 to 38° C. Pulse 60. Stupor, vomiting. No pus in nose. External frontal sinus operation. Posterior wall of frontal sinus showed small perforation. Dura covered with granulations. Dura incised. Abscess in frontal lobe evacuated. One-half to two drams. Recovery.

Case 34.--Foster Kennedy: *Journal of Nervous and Mental Diseases,* 1911. Struck over right frontoparietal region with spent bullet. Bullet entered bone, but did not penetrate cranial cavity. Six months later, headache and irritability. Optic neuritis on left side. Slight left facial weakness. Cerebrospinal fluid normal. Wassermann reaction in fluid positive. No history of syphilis. Loss of smell on right side. Retrobulbar neuritis of right side. Gradually increasing left hemiplegia. Operation showed large abscess in frontal lobe. Recovery.

Case 35.—Ernest Sachs: *Journal of Nervous and Mental Diseases,* 1911. Boy, 4 years of age. Two weeks before admission, fell and struck on forehead. Dull and irritable. On day of admission, right arm and leg became suddenly paralyzed. Temperature 99 to 104° F. Lumbar puncture gave clear sterile fluid. Three days later, unconsciousness, and convulsions more marked on the right side. Vessels of left retina tortuous. Operation. Frontal lobe abscess.

Case 36.—W. Freudenthal: *International Journal of Surgery*, September, 1908. Miss J. S., 25 years of age. Acute right frontal sinusitis. Headache and nasal discharge. Edema of right eye. Killian operation. Posterior wall of sinus eroded. Dura bulging. Dura incised. Two ounces of pus evacuated. Following operation several attacks of convulsions on left side. Death several days later. No autopsy.

Case 37.—Youl: *Lancet*, 1847; abstracted by Holt, *Archives of Pediatrics*, 1898. Child, 8 months old. Received wound in temporal region with some sharp body which healed. Two weeks later, vomiting and general convulsions. Died in one hour. Autopsy showed abscess containing two ounces of pus, in frontal lobe, at point opposite scar of external wound. Surrounding brain tissue much disorganized. Abscess communicated with lateral ventricle.

Case 38.—Wyss: *Jahrb. f. Kinderheilk.*, IV; abstracted by Holt in *Archives of Pediatrics*. Boy, 10½ months old. Fell several feet and struck on head. Four days passed before first symptoms occurred. Sudden onset with crying and severe pain at night, followed in ten hours by twitching of right arm and leg. Eight days later, paralysis of right arm and leg. Fever. Circumference of head increased. Six weeks after onset, coma. Pupils equally dilated. Six to eight convulsions daily, during one of which the child died. Autopsy showed left hemisphere larger than right, owing to an enormous abscess involving almost the entire frontal lobe. No communication with ventricles, which were distended with clear serum.

Case 39.—Koehler: *Irrenfreund*, Heilbrunn, 1879, Vol. XXI; abstracted by Holt in *Archives of Pediatrics*, 1898. Boy, 18 months old. Fell, striking head against a file, which penetrated frontal bone. Four weeks later scar was inflamed, and piece of file, one inch long, was removed. Later, vomiting and coma. Small swelling appeared at aperture in frontal bone, which pulsated and was easily reduced. Pus removed by aspiration and patient improved. Coma again developed, but upon aspiration no pus. Tremor of left half of body. Death in coma. Postmortem: Abscess size of egg in left frontal lobe, communicating with opening in frontal bone and with left lateral ventricle. Pus had penetrated through the foramen of Munro into right ventricle. Third and fourth ventricles also contained pus.

Case 40.—Borelius: *Jahrb. f. Kinderheilk.,* Vol. XXXII; abstracted by Holt in *Archives of Pediatrics,* 1898. Boy, 4 years old. Fell upon splinter of wood, which punctured upper eyelid. One week later, lid swollen and epileptiform seizures and other cerebral symptoms. No paralysis, but twitching was more marked in left arm and leg. Deep incision was made in upper eyelid, and large amount of pus evacuated. Probe passed through roof of orbit into frontal lobe. Drainage tube inserted, and patient recovered. Discharged in one month.

Case 41.—Zdekauer: *Jahrb. f. Kinderheilk.,* 1893, Vol. XXXV; abstracted by Holt in *Archives of Pediatrics,* 1898. Boy, 3¾ years old. Fell upon nail, which pentrated left side of head. Five days later fever, right hemiplegia and left-sided ptosis. Temperature 39° C., pulse 45. Over left temporal bone, wound from which pus discharged. Operation performed, during which dura was found to be fluctuating. Puncture of dura released two ounces of green foul-smelling pus. Drainage tube inserted. Recovery, except for paresis of left hand.

Case 42.—Reiner: *Jahrb. f. Kinderheilk.,* XI; abstracted by Holt, in *Archives of Pediatrics,* 1898. Male, 3 years old. Rickets. Cerebral symptoms of six weeks' duration. Bone disease for two and a half months. Then vomiting, fever and convulsions. Headache of left side and anesthesia of left side of face. Diminished hearing on left side and paralysis of left sixth nerve. Convulsions and high fever, loss of speech and loss of consciousness. Cornea ulcerated and perforated. Death in ten days. Postmortem showed large abscesses occupying nearly whole of left frontal lobe. Turbid serum in ventricles. Purulent meningitis at convexity and on left side of base. Extensive necrosis of frontal bone. Dura was adherent here and greatly thickened.

Case 43.—Treitel: "Ueber Hirnabszesse nach Stirnhoehleneiterung." *Deutsche med. Wochenschr.,* 1898, Vol. 22. Female, 22 years of age. In March, 1894, nasal obstruction, purulent discharge from left side of nose and pain in forehead and cheek. Polypi were removed from left side of nose on several occasions, as well as middle turbinate. In December, 1894, severe headache on left side of forehead. External operation on frontal sinus. Posterior wall was rough.

Pain did not disappear after the operation. In April, 1895, she was operated by Dr. Rotter. He found dura exposed over an area of one cm. A larger exposure was made and the brain punctured. No pus was found. On May 20th, repeated convulsions. Hemiparesis of right side. Pulse normal. Pupils equal and reacted well. Eye grounds normal. On May 22d, repeated punctures of brain were made. No pus found. Brain hernia followed. After two weeks, portions of this became gangrenous. This was removed and base cauterized. About a dram of pus escaped. On August 6th, patient was discharged, although wound still secreted pus and the hemiparesis remained. On September 11th, she was readmitted and frontal sinus again operated on. In October, convulsions. In March, 1896, the ethmoids were removed. On March 21st, death during a convulsion. Postmortem: Over left frontal lobe dura adherent to brain. Large abscess in frontal lobe. Basal surface of frontal lobe hemorrhagic. Purulent basillar meningitis.

Case 44.—J. M. Lee: "Abscess of Frontal Lobe of Brain and Convulsions Due to Accidental Puncture of the Orbital Roof." *Homeopathic Eye, Ear and Throat Journal,* 1897, III. Male, 12 years of age. Fell on stub of weed, which penetrated right upper eyelid, and passed through sphenoidal fissure into frontal lobe of brain. Wound healed without medical treatment. Six months later, convulsions. Small suppurating point on lid incised, and finally a piece of the stub worked out. Wound healed. A year later spasms reappeared, and lasted three weeks. Cicatrix again opened, and piece of stub removed. He was not troubled for nearly a year, when convulsions returned, and lasted six months. A third incision revealed no foreign body. Spasms subsided again. A year later they reappeared. Eye grounds normal. A U-shaped flap was made on forehead. Two buttons of bone were removed by trephine. Dura bulged and showed a small opening through which seropus discharged. Dura was incised, and cavity was seen in frontal lobe. Drainage. Spasms disappeared in one week.

Case 45.—Hanot: *Archiv. Gen. de Med.,* Paris, 1890. Large abscess of frontal lobe, without paralysis or convulsions, giving during life the clinical picture of tubercular meningitis.

Case 46.—Wm. MacEwen: "The Pyogenic Infectious Diseases of the Brain and Spinal Cord."—(1) Boy, 11 years of age. Was admitted on May 9, 1889. Ten weeks before, he was struck by a stone on the forehead, an inch above the left eyebrow. The boy was unconscious for half an hour. The wound healed in three weeks. Beginning four days after the injury, there occurred headaches, which gradually increased in intensity. For six weeks past, somnolence and repeated attacks of vomiting. About a month before admission there occurred, after the vomiting, profuse perspiration. Pulse was slow and weak; respiration slow and irregular; temperature normal. Patellar reflexes were diminished. Pupils were unequal, the left the smaller. Reaction normal. Slight paresis of the left internal rectus. Bilateral optic neuritis. On May 17th, convulsions in both arms, but most marked in the right. Then pleurosthotonus to left side. Operation without narcosis. Incision through the scar disclosed carious bone. This was removed and an area of thickened dura was exposed. Dura was incised, showing pia adherent to it, and inflamed. An aspirating needle encountered pus in the frontal lobe an inch from the surface. Abscess opened, and decalcified bone drainage tube inserted. Immediately after operation there was an increase in rapidity of pulse and respiration. Consciousness returned. Gradual improvement of all symptoms.

Case 47.—(2) Boy, 11 years of age. Admitted in 1876. Two weeks before admission he fell and struck his forehead upon the floor. Six days later, headache. On admission, wound, one and one-half inches long, was seen above left eyebrow. Roughened bone could be felt at the bottom. Somnolence. Rectal temperature, 100° F., pulse 60 to 65. Six days later, vomiting. The following day, chills. In the evening, temperature 103° F. Five days later, i. e., twenty-six days after the injury, convulsions, beginning in the right side of the face and extending to the right arm and leg. This followed by paralysis of the right side, and aphasia. The aphasia lasted an hour, then disappeared. Paralysis improved after several hours. Diagnosis was made of abscess in third frontal convolution, between speech center and internal capsule. Operation was refused. Two days later, death. A partial postmortem was allowed. A plate of bone was removed from the temporal region, the upper margin of which corresponded to the third frontal convolution. An extradural exudate was disclosed, and hyperemia of the dura. The dura was incised and reflected, and the brain incised. At a depth of one inch from the surface an abscess was encountered.

Case 48.—R. T. Williamson: "A Case of Abscess in the Right Frontal Lobe of the Brain." *Medical Chronicle,* 1890-91. Male, 19 years of age. Admitted March 22, 1890. On Februay 18th, influenza with profuse nasal discharge and frontal and occipital headache. Mentality dull. This dullness became progressively more marked. Knee jerks absent. Optic neuritis in right eye. Temperature between normal and 106.4° F., antemortem. Death on April 4th. Postmortem: Large abscess in anterior part of right frontal lobe. A small amount of pus in right ethmoidal cells.

Case 49.—J. C. Renton: "Notes on a Case of Cerebral Abscess, Subsequent to Orbital Periostitis." *Glasgow Medical Journal,* 1886. Male, 12 years of age. Admitted October 17, 1884, for what appeared to be a right orbital cellulitis of ten days' duration. On October 22d, an incision was made along the inner side of the upper eyelid, with evacuation of pus. On October 28th, severe headache, pulse 64, temperature normal. Vomiting. On November 11th, spasms of left side, which soon became general and ended in death on the same day. Temperature was never higher than 99° F., being generally 97.6° F. Postmortem: Convolutions over right frontal lobe, slightly flattened. Right orbital plate necrosed. Dura inflamed over this area. Abscess in anterior half of right frontal lobe. Abscess cavity lined by distinct membrane. There was no direct communication between the pus from the orbit and the cerebral abscess.

Case 50.—J. S., male, 27 years of age, entered my service at the Manhattan Eye, Ear and Throat Hospital on November 10, 1912, suffering from slight deafness in the right ear, of seven years' duration; intermittent purulent discharge; pain in the right ear for two weeks; mastoid tender; drooping superior wall of canal. Mother died of apoplexy at the age of 52. The patient never had had any serious illness. Venereal disease denied. Occupation, glass blower and prize fighter.

On November 12th, under gas ether anesthesia, I performed a Schwartze-Stacke radical operation. The mastoid was partly pneumatic, partly eburnated, and contained much pus. The middle ear and the attic contained much granulation tissue, and the malleus and the incus were necrosed. The stapes was located and was not disturbed. Neither the dura nor the sinus was exposed. A flap was made and the wound sutured behind. Packed with sterile gauze.

The wound pursued an uneventful course, and he was transferred at the end of eight days to the out-patient department for dressings.

On November 28th, sixteen days after the operation, while being dressed in the clinic by a member of the house staff, the patient had a sudden gush of blood from the middle ear cavity. The cavity was quickly packed with iodoform gauze, but the bleeding was not controlled. The hemorrhage was profuse, flowing through the gauze packing in a stream off the chin. It was checked by Dr. Stucky, the house surgeon, who repacked firmly with iodoform gauze. The patient was placed in bed. He was weak and pale, but the pulse was of fair quality. Temperature remained below 100.° A full dressing was done on the fourth day after the hemorrhage, when the wound looked clean.

On the morning of the fifth day (December 4th), at 5:30 o'clock, the patient had a convulsion, lasting from five to six minutes. He was not seen by a doctor during this convulsion. The nurse described it as "severe," "the patient shaking all over, the head retracted, with frothing at the mouth and gripping at the bed clothes with both hands." The house surgeon arrived as the convulsion was over. He described the condition as follows: "Cold perspiration over the entire body. Both pupils markedly contracted, equal and reacted to light. The patient could not be aroused to answer questions. The pulse was full, strong, regular, 84; temperature, rectal, 102.° Respirations 34. A full dressing was done and the wound looked healthy."

Fifteen minutes later there was another convulsion, lasting for a few minutes. This was also over before the house surgeon could be summoned. At 9 a. m., the temperature was 99,° respirations 22, pulse 112. At 3 p. m., the temperature was 100.8,° respirations 24, pulse 116. Blood count: Leucocytes 15,000; polymorphonuclear leucocytes, 89 per cent; small lymphocytes, 7 per cent; large lymphocytes, 4 per cent. Wassermann positive on December 10th. Blood culture negative. Spinal fluid under high pressure, but clear and "non-meningitic," according to chemical tests by Drs. Kopetzky and Callison. Color clear, copper reducing substance present. Lactic acid, marked reaction. Noguchi test weakly positive. Hydrochloric acid precipitation test negative. No polymorphonuclear leucocytes in stained smears. Culture negative. Urine negative, except for faint trace of albumin. Eyes, fundi normal. (Drs. Webster, Thomson and Van Fleet.) After the convulsions, the patient lay with eyes closed and drawn expression, but usually quiet. He complained only of headache, and if asked how he felt he replied, "All right, except for a dull headache." Asked to localize the headache, he said that it was "over the whole right side of the head and forehead." The headache persisted and the patient very gradually became stuporous. The whole right side of the head became tender to percussion. He did not answer questions promptly, but up to two hours before he became comatose answered intelligently. He remembered his name, age, address, and could give a good history of his trouble. The temperature never went higher than 102,° nor lower than 99.4.° The pulse was never lower than 80, until December 10th, when it varied between 60 and 78—always full and regular. On December 6th, Kernig was negative, Babinsky negative, no ankle clonus, no stiff neck. On December 10th, Kernig positive, Babinsky positive, the neck stiff, no ankle clonus. On December 11th, Kernig positive, Babinsky positive, ankle clonus very marked and equal, and neck stiff. The patient had an attack of typical Jacksonian epilepsy, beginning in the toes of the left foot and extending over the whole left side.

The patient was at once placed on the table, and the middle cerebral fossa was exposed by removing the tegmen. Incision through the dura gave two to three ounces of thick, creamy, foul pus, which seemed to flow from the anterior limit of the incision. The brain tissue protruded rapidly until a hernia of the brain filled the bone wound. Examination of the brain substance with a knife in all directions, failed to locate any deep abscess. A cigarette drain was inserted. After the operation, temperature was 102,° pulse 124, respirations 22. Intravenous saline infusion, on the table. The patient never regained consciousness. The temperature remained between 102° and 103,° until December 15th, just before his death.

Autopsy by Drs. Braun and Friesner and Stucky. There was a large subdural abscess over the right hemisphere, extending posteriorly to the posterior limit of the parietal lobe. There was a large abscess (See Fig. 1), the size of a hen's egg, thick-walled, full of greenish-yellow, foul pus, occupying most of the right frontal lobe; this abscess had burst and drained into the subdural space and probably was the source of the subdural abscess. The accessory sinuses of the nose were absolutely normal. The internal carotid artery contained a clot and was eroded at the knee, where there was a dehiscence of the bone. (See Fig. 2.)

This case presents several points of great interest to me.

First.—There was no doubt, early in the case, that there was intracranial pressure; and early exploration of the brain was not undertaken because there was no localizing symptom. The attack of Jacksonian epilepsy was the first localizing symptom.

Second.—Exposure of the dura through perfectly healthy bone of the tegmen of the antrum and the attic revealed what at the time of operation was supposed to be a superficial absess in the temporosphenoidal lobe of the brain.

Third.—The presence of a large deep abscess was strongly suspected, because of the rapid formation of the large hernia of the brain, and the brain was thoroughly, though fruitlessly, explored.

Fourth.—The finding of an unsuspected abscess in the frontal lobe, the fact that it had burst, and the fact that the pus from it was the cause of the subdural abscess.

Fifth.—The pus from the abscess showed no growth after forty-eight hours on Loeffler's blood serum.

Sixth.—The presence of a dehiscence in the bony canal of the internal carotid artery, and the presence of a perforation through the walls of the carotid artery at the site of the dehiscence in the bone.

Seventh.—The occurrence of a severe hemorrhage from the internal carotid fourteen days after operation.

This case may not have been of otitic origin, but I believe it to have been. It seems reasonable to suppose that the dehiscence in the bone of the carotid canal allowed of an erosion and infection of the walls of the artery, and that the abscess was the direct result.

One of the most constant localizing symptoms of frontal lobe abscess is hemiplegia. In the case under discussion this symptom was undoubtedly due to the extension of the subdural abscess over to the motor area. In some of the other cases reported, the hemiplegia was due either to the abscess involving the internal capsule, to the accompanying encephalitis which involved the internal capsule, or to pressure of the abscess upon the motor area.

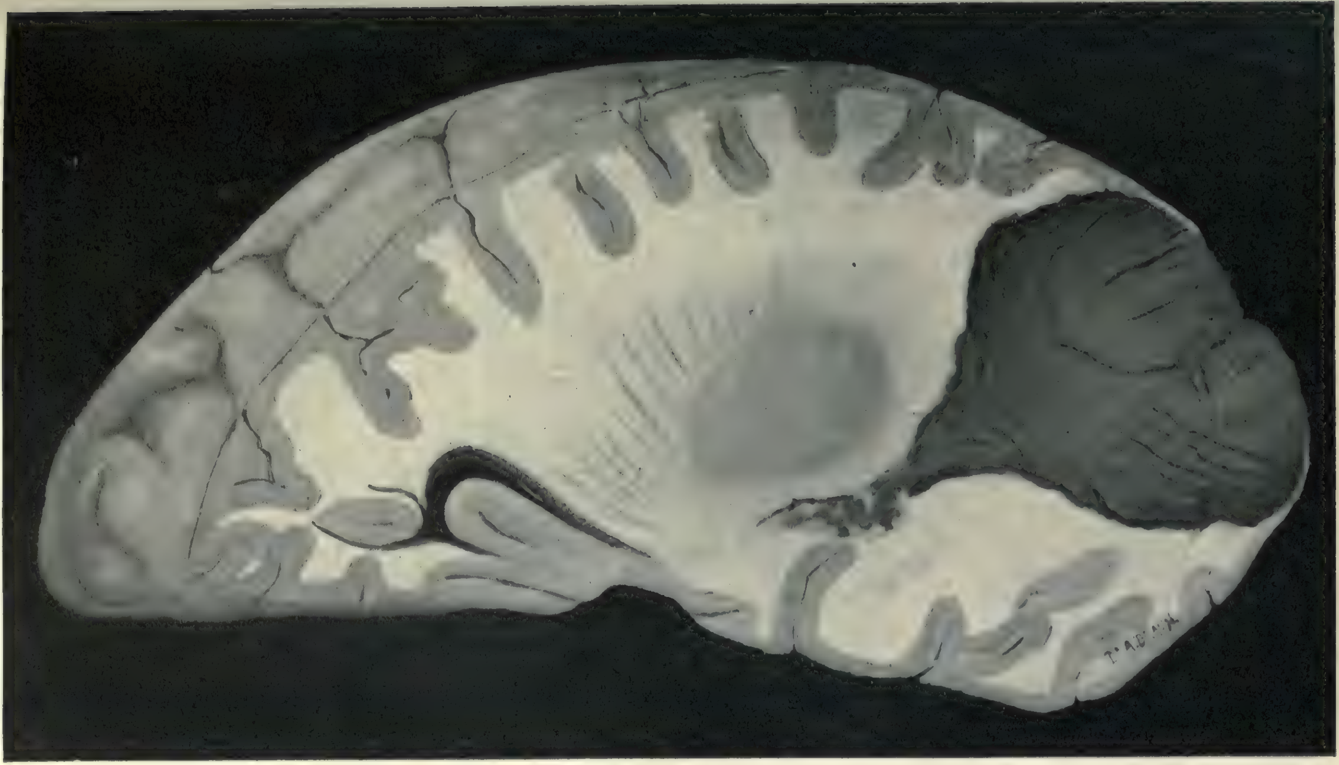


FIGURE I.

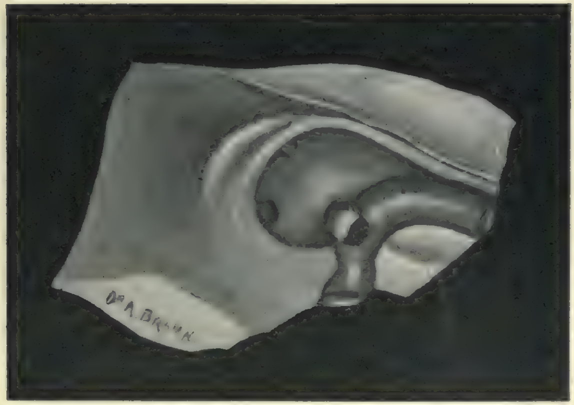


FIGURE II.

XXIV.

REPORT OF THE COMMITTEE APPOINTED TO CONSIDER THE BEST METHODS TO BE FOLLOWED IN THE TEACHING OF OTOLARYNGOLOGY IN UNDERGRADUATE AND POSTGRADUATE SCHOOLS.*

BY

D. J. Gibb Wishart, M. D., Toronto.
S. MacCuen Smith, M. D., Philadelphia.
Chas. W. Richardson, M. D., Philadelphia.

The subject assigned to our deliberation naturally divides itself into two distinct parts, and yet in the final analysis the postgraduate teaching is closely dependent upon the thoroughness of the undergraduate work.

FIRST.—"THE TEACHING OF OTOLARYNGOLOGY TO UNDERGRADUATES."

In order to ascertain what methods are followed and what views are held in the various leading universities of this continent upon this question, your committee submitted a series of questions to the professors of otolaryngology in forty of the leading institutions, the list of these being compiled from the Carnegie Foundation Bulletin No. 4, entitled "Medical Education in the United States and Canada."

These questions were as follows:

1. Should the "course of instruction" be extended over one or two years?
2. How many hours should be devoted to this "course of instruction"?
3. What proportion should be clinical and what didactic?
4. In what order should these be given?
5. What should be the limits of such a "course of instruction," having in view the necessities of the general practition-

---

*Adopted by the American Laryngological, Rhinological and Otological Society, May, 1913.

er, and the proper balance of the subjects of a medical curriculum?

6. Should "operations" form part of such a "course of instruction"?

7. Should a separate examination in otolaryngology form an integral part of the final examination?

N. B.—In answering the above questions, which are intended to ascertain the personal views of those responsible for otolaryngology in the leading medical colleges of the United States and Canada, it is desirable to indicate briefly also to what extent these personal views are carried out in the institutions with which the writer is connected.

Replies were obtained from some thirty-one institutions, including over twenty states and provinces, so that the opinions elicited may be considered fairly representative. In perusing these reports your committee was agreeably surprised to find so little divergence in either practice or opinion as the replies revealed, and have therefore the greater confidence in submitting the resolutions which form their conclusions.

We regret that we were unable to elicit satisfactory information as to the amount of "individual instruction" given to each student. Where the number in the classes are considerable (and if possible that number should never exceed six students), as Mygind[1] points out, "the student is apt to acquire superficiality in the examination of the patient." This fault is undoubtedly in many cases due to the teacher not having sufficient time to take each pupil individually and teach him all the details he should observe."

We note also that the idea still lingers in the minds of some of our leading instructors, that didactic lectures form an adequate means of instruction. About one-fourth of those reporting asked for equality between didactic and clinical instruction. Surely this cannot be right. The regions involved in otolaryngology are such as lend themselves least readily to demonstration by lectures, or even by lecture-clinics. It is only in so far as the student receives "individual instruction" on the living subject, that he can acquire an adequate knowledge of his subject in the limited time which can be spared in the overcrowded medical curriculum.

This "individual instruction" requires time, patience and competent assistants; but these are well worth the expense involved, as by properly instructing the student of medicine in otolaryngology, we will do much to avoid the conditions which now lead to the foisting of vast numbers of what we may style pseudospecialists upon the public. To quote Mygind[2] again: "A few cases examined and followed thoroughly teach the student to observe, and give him better experience than a great number of cases examined superficially."

"Individual instruction," as pointed out above, demands competent assistant instructors, but the governing boards of our universities must be compelled to realize the absolute necessity which exists for the proper balancing of the medical curriculum, so as to turn out all around general practitioners, and must provide the equipment therefor.

Another point which your committee deem worthy of criticism in dealing with these reports is the scope of the instruction with regard to text books. Such a course should get as far away from the text book as possible. We cannot in the least agree with the gentleman who reported in answer to question five, "that the contents of the required text book on these subjects should be covered, *chiefly by recitations*." (Note the italics.) The student should be led to read up the cases he sees in one of the many excellent text books which are at his command, but to cover a text book by recitation is simply to waste time, and must lead to superficiality.

Every student of medicine has a right to be graduated primarily as a general practitioner. The first task of every general practitioner is diagnosis. No amount of text book recitation will develop the ability to diagnose correctly in otolaryngology, unless there go beforehand the visual knowledge of the normal as contrasted with the abnormal, the education of the eyes, ears and fingers. This education means actual work upon the patient, and the "course of instruction" should therefore be almost wholly clinical.

As regards the order in which instruction should be given, some of the teachers feel strongly that any didactic instruction should be given as the finale, to enable the student to coordinate his observations and place them in related groups.

By question five we hoped to elicit a sufficient answer to a difficult problem. With the daily advance of medical science, new subjects are with loud voices demanding admission above course, except that in so far as may be possible, each student should be permitted to assist at the performance of the simpler varieties, that he may become practically acquainted with the methods of procedure and the objects sought.

Seventh.—That a special separate examination in otolaryngology, preferably clinical, conducted by the professor of otolaryngology, should form part of the final examination in medicine of every university and every licensing body.

Eighth.—That a copy of these resolutions be forwarded to the medical faculty of every university and college of medicine, as well as to every state or provincial examining board in the United States and Canada.

SECOND.—THE TEACHING OF OTOLARYNGOLOGY TO POSTGRADUATES.

The second part of our report concerns postgraduate instructions in otolaryngology, and while the former was aimed at the production of the well rounded general practitioner, this must endeavor to achieve the evolution of the scientific specialist.

The subject is much to the fore on this continent and elsewhere. Last year the *Journal of Laryngology, Rhinology and Otology* published an able series of articles upon the education of the specialist in throat, nose, and ear disease, in Denmark, Germany, Italy, Austro-Hungary, and France. In Britain, it formed a topic of general discussion at the Liverpool meeting of the British Medical Association. On this continent it has led to the publication of recent papers by Shambaugh,[4] Ellett,[5] Cragin[6] and others, and formed the basis of an interesting discussion before this society two years ago, a discussion which led to the formation of the committee now reporting. A pronouncement by this important organization will therefore be quite apropos at this serious juncture, for we must not forget that with privilege comes responsibility, and this society, whose membership is so extensive and whose influence so widespread, should, noblesse oblige, lead, not follow, in the making of an intelligent public opinion.

In opening the discussion of the question before the British Medical Association, Mygind[7] of Copenhagen stated that "Although there is no regular system of education of the specialist in otolaryngology in Germany or Austria, we may divide the practical education into three stages, through which the specialist generally passes, both in these two countries and in most other continental countries.

1.—The first stage: The preliminary education which each student received at the university. It is compulsory in most continental countries and extends over a period of one term.

2.—The second stage is represented by the education which many future specialists receive, after having left the university, at one of the numerous postgraduate courses which nearly every university and many hospitals offer, and which are given either by professors or by private docents attached to the university. In Austria they are, according to Frey, divided into six different kinds: (a) Clinical diagnosis and treatment; (b) functional tests; (c) bedside work; (d) surgical practice on the cadaver; (e) practical treatment and minor operations in out-patient department; (f) demonstrations and lectures on normal and pathologic anatomy, histology, and physiology, to which quite lately has been added (g) pathology and diagnosis of labyrinthine diseases.

3.—The third stage is the education the future specialist receives as assistant at a special clinic attached generally to a university or a general hospital.

The third stage is undoubtedly the backbone of the whole education of the specialist, and it is only a rare exception that a medical man on the continent nowadays is universally recognized as a specialist without having been for some time assistant at a public clinic. In Germany and Austria, and also in some other continental countries, so much stress is laid upon this part of the education that one often meets specialists of a considerable scientific reputation remaining for a considerable number of years assistants at a special clinic, in order to stand a good chance when a chair in otolaryngology is vacant at a university. After having been appointed an assistant for a considerable period at a special clinic, and after having given proofs of scientific ability—in Germany an officially approved dissertation must be printed—the specialist may have in Germany, Austria, and Italy the title of privat docent conferred upon him by the university.

This gives him the right of teaching in the university and enrolls him in that high but by no means numerous class of specialists from whom the professors are chosen."

Up to the present on this continent there is no recognized portal to the specialty. On the other hand, the gates may be said to be many, and yet, "mirabile dictu," our towns and villages and even our cities are filled with "specialists" who have entered by no gate whatever, but have simply "climbed over the wall," and are to some extent, at least, to be considered merely as "thieves and robbers."

The house surgeon attached for three months to the otolaryngologic service of a hospital, the general practitioner who derives his knowledge of the subject from a six weeks' course in a postgraduate school, and the man who takes a run in Europe immediately after graduation, alike think themselves worthy to be ranked as specialists.

As long ago as 1892, Osler[8] pointed out that there is "serious danger in the attempt to manufacture rapidly a highly complex structure from ill seasoned material."

As Shambaugh says,[9] "there is a deplorable lack of proper standards in medical education, and the specialties of the eye, ear, nose, and throat have had to carry a disproportionate share of these ill trained practitioners."

Again, as Amberg so clearly puts it, "From the standpoint of a well regulated and governed community, it represents anarchy, and anarchy is the absence of government, the state of society in which there is no supreme power."

It is time we had done with this farcical sort of preparation, if our specialty, worthy as it is of the best, is not to be dragged in the mire as a result of the ignorance of anatomy, diagnosis, and technic displayed by a very large proportion of the rank and file of those who now style themselves "specialists in diseases of the ear, nose and throat."

We all know the facts, and we deplore them. Now, what can be done to face and overcome the difficulties of the situation? We must first decide what constitutes the standard of proper training, then provide for its acquirement, and finally bring such influence to bear upon state legislatures as to secure the legal enactment that only specialists provided with this training may practice as such.

Those who have written upon this subject are agreed that not less than two years should be devoted to preparation for practice as a specialist. This should, however, be preceded by one or more years spent in general practice; or better, as house man in the medical and surgical services of a good hospital, at least half the time being devoted to surgery. The specialist course itself must embrace highly specialized studies in the anatomy, physiology, embryology, pathology, physics, and therapeutics, which bear upon the subject, operation on the cadaver, etc.

The time required for this course will occupy at least six months. In addition the candidate must subsequently serve as a resident assistant in a special hospital or in a similar position in the special service of a large general hospital, for a period of not less than eighteen months, and to provide sufficiency of experience, the special clinic should work daily, and should have at least fifteen beds assigned to its use. In this connection it is well to insist that during the last six months of his service the candidate shall act as a senior assistant in the major operations and personally perform those less important.

Where shall such training be provided? Without doubt the scientific part must be placed solely under the control of the universities, for these are the proper bodies to provide postgraduate instruction. In as much as the universities are amenable to the public opinion of such bodies as our own, and as they stand for increasingly higher ideals, there is a guarantee that the instruction provided would be thoroughly scientific and comprehensive.

Not every university could at present undertake such postgraduate instruction, but a few judiciously selected centers could be found even now, where such courses as we suggest could be given.

With regard to the hospital training of the candidates we are not so limited. Several first-class special hospitals exist whose housemen are in demand by institutions which cannot now train their specialist juniors.

The terms of service in these special hospitals are, so far as we know, never less than eighteen months.

In the special services of the larger general hospitals it is becoming increasingly common for the house surgeon in otolaryngology to be obliged to serve for eighteen months, to confine himself to these subjects only, and to be selected from applicants who have been several years out of college and who have decided to devote their future to otolaryngology.

It would not be difficult to draw up a schedule of the requirements which such institutions must fulfill, if their certificates of services rendered are to count in the qualifying of candidates.

It would not be necessary to confine the candidate to such positions as he could find on this continent, and in any event there might easily be arranged provision for the candidate pursuing further study in Europe.

To add a fitting coping stone to the structure whose erection we are considering, the candidate should finally be compelled to present himself to one of the universities supplying the postgraduate instruction indicated above for examination on the work embraced in the entire course, and the successful candidate should be given a degree—that of Ph. D. (otolaryngology), has been suggested in several quarters.

In sketching the above course we have drawn nothing fanciful, for at the present time every man who is desirous of placing himself at the top of the ladder in our specialty, in one way or another pursues such a course of study, but often finds with regret that he has not been wisely advised in his choice of work, and places to work in, and there is no arrangement for the examination anywhere, except that indicated in the above quotation from Mygind, viz., in Germany.

The final step, that of securing legislative measures, would of necessity be difficult to attain, but public opinion will ere long demand that we be protected from the incompetent specialist as we are now from the ill trained dentist.

The program sketched above takes no account of the postgraduate school as at present organized, nor need it do so. The case against the present condition in postgraduate teaching has been ably stated by Birkett,[10] and need not be repeated here.

By no reconstruction which we know could we hope to secure any control of institutions which are simply, after all, incorporated for the purpose of dividends. They came into existence to fill a need produced by the poor character of the instruction in general medicine given by many of the schools of medicine, many of which were themselves proprietary, and. as Shambaugh has already said, "when the undergraduate instruction in medicine has been thoroughly done, the elementary work in the specialties will not require the subsequent patching, which is all that the existing postgraduate schools are prepared to do." The Carnegie Report has already effected a marvelous change through the public opinion its revelations have aroused and the proprietary school is doomed.

With regard to postgraduate teaching in otolaryngology, your committee beg to recommend—

First.—That the time has arrived to standardize the degree of scientific attainment and of clinical training which shall qualify those who wish to begin the practice of otolaryngology as a specialty.

Second.—That the report of this committee now submitted forms a suitable basis for such standardization.

Third.—That a committee representing this society be appointed to work out the details.

Fourth.—That the other associations of otolaryngologists on this continent, viz.,, The American Medical Association, The Academy of Ophthalmology, Laryngology and Rhinology, The American Laryngological Society, The American Otological Society, be and are hereby invited to appoint representatives to act with the above committee from this society, so that the action finally taken shall represent to the fullest extent the consensus of opinion of American and Canadian otolaryngology.

REFERENCES.

1. British Medical Journal, August 24, 1912.
2. Ibid.
3. Transactions American Laryngological, Rhinological and Otological Society, 1911.
4. Journal American Medical Association, September, 1912.
5. Southern Medical Journal, January, 1911.
6. Medical Record, Vol. 80.
7. British Medical Journal, August 24, 1912.
8. Boston Medical and Surgical Journal, 1892.
9. Journal American Medical Association, September 21, 1912.
10. British Medical Journal, August 24, 1912.

XXV.

SUSPENSION LARYNGOSCOPY WITH DEMONSTRATION OF METHOD.*

By Wolff Freudenthal, M. D.,

New York.

It is not so long ago that we thought inspection of the air passages was a closed chapter and that nothing more was to be accomplished in that direction. Then Killian introduced bronchoscopy, and this was followed by other new ideas. I need only mention the names of Mosher, Jackson, von Gyorgyai, Hays, Yankauer, Beck, and Edgar Holmes of Boston, to bring to your minds a great deal that has been done of late. But laryngology is advancing rapidly, and today again we have something entirely new, i. e., suspension laryngoscopy.

This latest discovery of Killian's is not only very interesting, but his method is of great practical value in many cases, and it has been tried by the writer in a series of experiments which have extended over nine months. In the first publication† on suspension laryngoscopy, its advantages and shortcomings were defined, and after larger experience we hold practically the same views.

It is unnecessary to describe the method just demonstrated in detail, as you have seen it today and can find the full account in the publication mentioned. Permit me, however, to give some additional points which may prove useful.

Besides the necessary instruments, the first thing required is good illumination. The best for operative work, and the only kind advisable, is a good Kirstein headlight. For other work, a small lamp (or two) attached sideways may be helpful. The second requisite is a room that can be darkened

---

*Read before the nineteenth annual meeting of the American Laryngological, Rhinological and Otological Society, held at Washington, D. C., May 8, 9, 10, 1913.

†Wolff Freudenthal: Personal Observations with Suspension Laryngoscopy. Medical Record, February 22, 1913.

(absolute darkness is unnecessary); and the third, an assistant with some experience in handling the apparatus. Trivial as these things seem to be, they are very important. The writer speaks from experience. Once, on being called to a general hospital to remove a growth from the larynx, a total failure resulted, owing to the lack of proper facilities. At another time the assistant was entirely at fault, as he manipulated the handle so roughly that he irritated the patient, who was of a very nervous temperament, and nothing could be accomplished under local anesthesia.

Only once a slight accident occurred, which I should like to mention: M. S., a short, rather heavy-set young man, twenty-five years of age, was curetted under suspension laryngoscopy on account of an ulceration of the posterior laryngeal wall (incipient tuberculosis of the lungs). The procedure took the usual course, but immediately after the operation the whole appearance of his face changed; it became swollen, highly red, and the whole forehead was bloodshot. He suffered no discomfort, however, and I ordered simple rest and codein. The next day there was not much change. In the pharynx there were still enlarged arteries on the right lateral wall and on the left tonsil. A few days later everything appeared perfectly normal.

Another point to be mentioned relates to the epiglottis. Years ago, when Kirstein first described his instruments for autoscopy, I noticed that the epiglottis in many instances could not be kept down with his spatula. At that time an attempt was made by me to overcome this difficulty by means of hooks which could be pushed over the epiglottis, these being made by Tiemann & Company. Later, however, this plan was forgotten, to be recalled only when the necessity for holding up the epiglottis again presented itself. The writer then resorted to the same device (see Figure), which consists of a silver wire fastened in the groove of the spatula at B and C, and ending in two thickened points, D and E. These can be pushed forward, and when they are in that position the epiglottis will remain in situ. By means of the hook A we can bring the wire forward or backward. The portion A—B can be turned to one side, if it is in the way. In the cases so far examined, this modification has worked very well. Since writing this I have noticed that Albrecht of Berlin had a similar idea, and published his modification of a tongue holder in the *Berlin. klin. Wochensch.*, p. 2092, 1912. I have not seen his instrument, as it could not be obtained in New York.

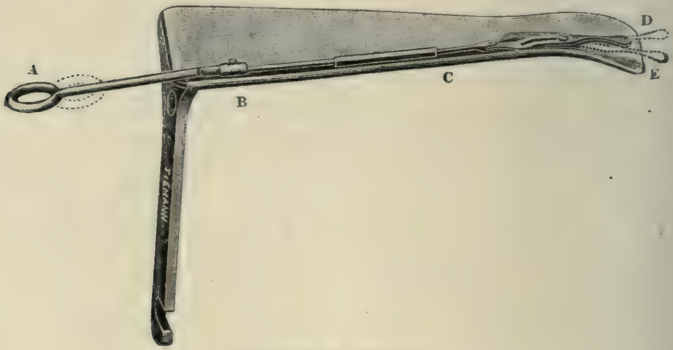
Permit me to give you the details of some of the cases handled by me, which may be divided into several classes.

I.—TUBERCULOUS CASES.

These have been the most instructive, but only those will be mentioned which were of special interest to me.

G. H., a sailor, aged 40, was referred to me in September, 1912, on account of hoarseness which had come on gradually during the last ten weeks. No pain, no cough, but a heavy feeling in his chest.

The larynx showed an irregular infiltration of the left vocal cord and a thickening of the entire right one, which was immovable. Quite a large ulceration was seen on the posterior wall. Examination of the chest revealed only the presence of a bronchitis; no tubercle bacilli. In spite of the fact that the lungs showed no positive evidence of any tuberculous affection, I took the case to be one of tuberculosis.



Epiglottis Holder.

On October 18th, several pieces from the vocal cord were removed, and the ulcerated surface was curetted under suspension laryngoscopy. The immediate result was satisfactory. The ulcer healed and patient could breathe easier. Within a week he left for a country place. At the beginning of December, 1912, he returned, as he did not feel comfortable. On

December 9th, the septum for the first time showed the presence of a few tubercle bacilli. There were some new ulcerations, which were treated with orthoform emulsion. The pain disappeared, but not the ulcers. These were curetted again under suspension on the 20th of December, and again the patient felt so much better that he left for the mountains.

In February, 1913, he returned to the city with some dyspnea. Sputum positive, but chest findings almost the same as in the beginning. The larynx, however, had changed surprisingly. There were irregular masses springing up from both vocal cords; the arytenoids were markedly swollen, so that very little of the posterior wall was visible, and little space was left for breathing. Patient did not care to undergo another operation, and was kept under observation, the dyspnea growing steadily worse.

March 17th.—The dyspnea was so marked that I came very near doing a tracheotomy in my office, but finally succeeded in getting him to the hospital. There I removed five large pieces from the larynx under suspension laryngoscopy, local anesthesia being employed. The bleeding during and after the operation was minimal, in spite of very extensive removal of tissue. I mention this merely on account of a remark made by Dr. Schoetz in the Berlin Laryngological Society. (Of course, this does not mean that the possibility of hemorrhage in tuberculosis of the larynx is excluded entirely, but only that we need not be afraid to operate on such cases.)

The further history of the case proved that we were right in our deductions. After the operation the patient was put to bed and given ice externally and by mouth. All the instruments required for a tracheotomy were placed next to his bed, in case of immediate necessity. His breathing was better than before the operation, but about six hours later it again became quite bad, due, undoubtedly, to edema of the larynx, which, however, subsided gradually. The next day, when I examined him, a good deal of edema was still present, as well as some dyspnea and dysphagia. The day following (March 19th) the picture had changed much for the better. The edema had largely subsided, he could breathe almost naturally, and felt entirely different. The dysphagia, however, was marked. Orthoform emulsion was applied.

March 24th.—No appetite. Patient felt like vomiting all the time. There was a false membrane in the larynx and a great deal of discharge from that source. Respiration free.

March 27th.—Fulguration was applied to hasten the process of healing, with the result that the edema again appeared next day, together with dyspnea. This subsided after application of cocain and adrenalin, but soon recurred. Examination showed a dislocation of the left arytenoid, which was flattened and lower than the right one.

The dyspnea became so marked that on March 31st I removed part of the overhanging tissue from the right vocal cord, in the presence of Dr. Layman of Indianapolis and other colleagues. After that the breathing remained free, and about two weeks later the patient again left the city.

In reviewing the history of this remarkable case, you will find several points of interest. First: The case presented the clinical picture of advanced laryngeal tuberculosis at a time when the lungs were very little or not at all involved. Second: After we had once curetted the larynx, infiltrations sprang up so rapidly that tracheotomy became imminent. But I refrained from that operation and instead removed five large masses from the larynx. I did not know that one could with impunity resort to such an extensive excision until in this case I was simply forced to do so in order to remove as far as possible all diseased tissue, and also to give breathing space.

You will naturally ask why tracheotomy was not done, which is so much simpler. To this I must answer that I was ready to perform this operation in the hospital whenever it should become necessary, but I tried to avoid it if possible. What would be gained by it in such a case? Only the danger of immediate suffocation would be averted; but afterward, what then? We would have had to resort either to a laryngofissure —and the mortality of that operation is very high indeed— or remove the enormous masses later with the aid of suspension laryngoscopy, and that probably would have been more difficult with the trachea opened. So, after careful deliberation, it was thought best to proceed as we did. The patient has gained free breathing space without having to undergo a tracheotomy, with the eventual necessity of having to wear a tube for a long period. He has now every chance for improvement.

Another case which is remarkable for the ease by which a cure was accomplished is that of a tailor, aged 30. He had suffered from tuberculosis for the last three years. He had been for about a year in Denver, Colorado, where his pulmonary condition was practically arrested. He looked well and strong, and the only thing that annoyed him was the larynx. On account of his hoarseness he could not get a position, and the irritation in his throat made him cough. Quite a good-sized interarytenoid growth was visible, which was apparently the sole cause of his trouble. In several clinics attempts at removal had been made, but they were unsuccessful. The writer had the same experience at his first attempt. When the patient was placed under suspension laryngoscopy, quite a large piece was removed, but afterwards we found that a good deal more had been left and we had to operate twice again, the last time in the presence of Dr. J. Solis-Cohen of Philadelphia, on April 16, 1913. The patient felt much better and soon withdrew from treatment. I believe him to be cured.

It must be added here that as a rule large masses of tissue are not removed at one sitting, as we do not keep such patients in the hospital overnight and edema of the larynx is apt to set in. It is only where there is no danger that we remove all that is necessary at once, even very large masses.

Whether this case and the one previously referred to remained cured or not, is immaterial so far as the method is concerned. For the time being the first patient's life has been saved, and the last patient's growth removed, thus giving them the best chances for recovery.

In this connection I may be permitted to give the further history of two cases reported before.

Mrs. Z., a very nervous and excitable woman, was suffering from tuberculosis of the lungs and ulcerations of the epiglottis and left vocal cord. She was curetted on November 8, 1912, as thoroughly as possible. The dysphagia disappeared to a great extent afterwards, and if it were not for the pain from the ulceration of the epiglottis—that could not be reached—she would have felt much better.

On February 16, 1913, the following note was made in my book: Patient had been treated for several weeks after the curettage according to the usual methods, and now she can eat and swallow like any healthy person. She had not come for treatment for the last four weeks, and now has pain in the chest—slight pleurisy.

March 12, 1913.—Status idem. She only came to ask whether she should take the serum treatment so much talked about. Larynx in good condition.

April 15, 1913.—She again has dysphagia, is unable to come to my office, and is being treated by a local physician. There is no doubt in my mind that the ulcerations have recurred and that she ought to be curetted again thoroughly under suspension laryngoscopy.

M. F. had suffered from tuberculosis for about eight years and had a large mass of tuberculous infiltration, with an ulceration on the posterior wall of the larynx. Both arytenoids seemed enlarged (perichondritis). He had been seen by many colleagues of this city. The writer also had had him under treatment for quite a time, but it was impossible to get rid of the infiltrations. Even fulguration did not exert any beneficial effect, as it was not well tolerated by the patient. Since his pulmonary condition did not bother him, he was naturally desirous of having his throat cured. On November 15, 1912, in the presence of a number of laryngologists who attended the Clinical Congress of Surgeons, quite a large piece was removed from the larynx. Part of the arytenoid had been caught and removed at the same time, but not the entire mass of granulations. Postoperative pain was very slight; no dyspnea. A false membrane soon formed over the site of operation, but soon disappeared. On December 20, 1912, the whole mass was curetted thoroughly, with very satisfactory result.

On February 2nd he had to be curetted again, and what followed in this instance was similar to the condition observed in the case reported by Halle to the Berlin Laryngological Society (*Berlin. klin. Woch.*, p. 321, 1913), viz., the picture of a paralysis of both postici muscles. It is evident that no paralysis of central origin was present, but that by the curettage of the posterior laryngeal wall scars had formed which brought the vocal cords in adductor position. Yet he has no dyspnea and feels quite comfortable, so that no other treatment is indicated.

It seems to us that through suspension laryngoscopy an extensive field has been opened for thorough and radical work in laryngeal tuberculosis. We are thus enabled to curette all ulcerations thoroughly, with the exception of those of the epiglottis, and we are furthermore in a position to remove the diseased tissues down to the healthy parts. This, for the first time, seems like real surgery of the larynx. How far we may go, the future will have to show us.

II.—ABSCESS FORMATIONS.

As mentioned very briefly in my first article, these cases offer a great opportunity for suspension laryngoscopy, especially those of abscess in the larynx. A case that occurred in my practice several years ago is still fresh in my memory. A young woman who had a retropharyngeal abscess suddenly developed severe dyspnea, the phlegmon having extended into the larynx. Both arytenoids were seen to be much swollen, apparently occluding the entire lumen of the larynx. One arytenoid was incised and a good deal of pus was evacuated. When the other was incised, the patient suddenly collapsed and fell to the floor. A hurried tracheotomy saved her life for twenty-four hours. How much better would it have been to have such a patient under suspension laryngoscopy, with the head hanging down and the abscess draining downward by its own gravity. These cases are quite rare, but if ever there was a strict indication for a certain method, it is in such patients.

While writing this article I happened to see another case, the history of which may be of interest. A married woman, aged 27, was referred to me on February 22d, on account of hoarseness which had persisted for six months and was gradually getting worse. She had a slight cough, but no other complaint whatsoever. I made a diagnosis of pachydermia laryngis, and treated her accordingly. Later on she suffered from tonsillitis, both tonsils being very much swollen, and it turned out that she had had such attacks before and that she was subject to rheumatism. It was decided to enucleate the tonsils some time after the acute attack was over. But no sooner had the condition of the tonsils improved than a swelling of the left arytenoid set in. This was considered as a purely rheumatic arthritis. In spite of all medication, the discomfort (dysphagia and slight dyspnea) was very annoying. Three days later the other arytenoid cartilage became swollen, though to a lesser degree, and at the same time true blebs appeared on top of the left arytenoid. As the dyspnea had increased, it was naturally thought advisable to open the bleb.

Remembering my former experience in that direction, I put the patient under suspension laryngoscopy, and at once saw not only the bleb on the left side, but also another on the right side, just below the glottis, which I had not noticed before. Both were incised, and patient felt easier. A very energetic antirheumatic treatment reduced both arytenoids materially, and she left the hospital without dyspnea. (There was no pemphigus present.)

Even in abscess formation higher up in the pharynx, suspension laryngoscopy is occasionally indispensable, and perhaps may be universally adopted in the near future. Already some of our laryngologists no longer open a retropharyngeal abscess in young children in the upright position, but prefer to do it with the head hanging down. Accidents have happened in their clinics, and they have learned to be careful.

One case occurred not long ago which may be worth mentioning. A house surgeon in one of the larger hospitals of this city called up his visiting surgeon during the night, asking his permission to open a retropharyngeal abscess in a young child. Consent was readily given. Some time later the visiting surgeon was called up again and asked what should be done with the child, as it had not been breathing for the last forty-five minutes! (Tracheotomy had been done!) Undoubtedly there are more observations of such fatal results, which always occur from the pus clogging up the glottis. How different is the procedure under the suspension method. We had a chance of operating on two such cases in infants of sixteen and nineteen months respectively. In both instances the abscess was rather deep down, but showed up beautifully as soon as the child was under suspension. The whole extent of the abscess was easily seen and still more easily evacuated. The children had received only a few whiffs of ether.

This brings us to the question of

III.—TONSILLECTOMY.

Shall they all be done under suspension when general anesthesia is employed? While the writer is not prepared to give a definite answer to this question, it is his experience that in older children the operation can be done easily in this position, and in many cases it is preferable to the prone or the upright posture. In younger children,—that is, below the age of three or four years,—the mouth cannot be opened sufficiently wide to allow the use of the comparatively big tonsil instruments with ease and satisfaction. In older children it is simple.

If you do operate in that way, it is wise to introduce the spatula sideways and so fix it that its end is not in the median line, but points toward the tonsil to be operated on. In this way, the tongue is kept entirely out of the way. If narcosis is carried out satisfactorily, the advantages of this mode of operating on the tonsils are in many cases very noticeable. The writer has operated on about twenty children in this manner, and finds that the benefits derived are due to the fact that the blood flows downward into the nasopharynx and gets out of the way. We do not require any suction apparatus, and can finish the operation quickly and without much assistance, always provided that the anesthesia is complete.

IV.—PAPILLOMA OF THE LARYNX IN CHILDREN.

It would seem that these cases are especially adapted to suspension laryngoscopy, but the writer has not had the good fortune to come across such a case for the past year. He has seen, however, a number of .

V.—BENIGN NEOPLASMS OF THE LARYNX IN ADULTS.

These patients were absolutely unmanageable under cocain, and the suspension had to be done under general anesthesia. The first requisite in these cases is perfect anesthesia, and you are lost without that.

The following case, upon which I operated in conjunction with Dr. J. Weinstein, may be of interest:

S. E., aged 52, agent. Hoarse for the past three years. The doctor had several times removed pieces from a mass on the left vocal cord, which were always found to be benign. For the last two months patient had had some dyspnea, and the larynx showed a large mass filling nearly the entire left side. As there was no strict indication for removal of the entire larynx, we took out as much of the diseased tissue as we could get. This was done under general anesthesia. The patient had quite a little edema of the larynx following the operation, as well as dyspnea, but these disappeared, and I understand that he is now doing well. Examination of the tissues removed again proved to be benign in character.

In cases where it is necessary to operate on the anterior commissure, which is naturally the hardest to reach, the epiglottis has to be out of the way entirely. For that purpose it may be sufficient in some cases to use a longer spatula or the writer's spatula with the hooks, described above. If neither of these proves adequate to bring the anterior commissure into view, as will be the case in the majority of instances, then one assistant has to press down the larynx from the outside. This helps in many cases.

Dr. J. Solis-Cohen of Philadelphia recently suggested that with a long, flexible-handled, polished steel mirror, one centimeter in diameter, one could examine and operate upon the anterior walls of the larynx and trachea under suspension laryngoscopic examination. As I had in my possession a long-handled glass mirror which had been devised for the inspection of the vocal cords from below, I straightened it and tried Dr. Cohen's suggestion. I was able to see the anterior portion of the larynx, but it is doubtful whether another instrument to remove any neoplasm could be introduced simultaneously with the mirror. Yet this is a very interesting experiment and encourages future trial.

VI.—FOREIGN BODIES IN THE PHARYNX AND LARYNX.

Dr. E. D. Davis of London removed a pin from the posterior pharyngeal wall of an infant eleven months old, under chloroform. He found the great advantage of suspension to be in the comprehensive view obtained by this method, as compared with the small area visible by the esophagoscope (*Brit. Med. Jour.*, January 18, 1913, p. 115). The operation was apparently very easy.

It seems to us quite natural that foreign bodies in the larynx should be removed under suspension laryngoscopy, and that we ought to be able to reach them very easily without the least risk.

In conclusion, I would say that while suspension laryngoscopy is not going to revolutionize the entire domain of laryngology, nor replace the old established methods in the large majority of cases, it will be—if it is not already—of great benefit, both diagnostically and therapeutically. In the many cases where through its aid a clear view of the operative field is gained, it has proved of incomparable value.

XXVI.

THE TRAINING OF THE SPECIALIST.*

By Thos. J. Harris, M. D.,

New York.

The last half of the nineteenth century witnessed a marvelous transformation in all walks of business. New methods and new means, inspired by such master minds as that of Morgan, were in evidence on every side. This development and expansion were not limited to business alone. They were equally apparent in every department of science. It was said of Goethe that his was the last great mind able to grasp all knowledge and all literature. In this wonderful advance medicine has borne a conspicuous part. There was a time when a physician could hope to master the entire realm of medicine. This has all changed, and this change has largely taken place within the last fifty years. Most, if not all, of this progress, embracing discoveries which have revolutionized the conception and treatment of disease, is due to those who in the truest and broadest sense were specialists. Specialism in medicine and specialists have come to stay, like it or not as we may.

Among the earliest, if not the earliest, to attain to the dignity of being a specialty, was that of laryngology. Horace Greene, denounced and condemned by his contemporaries, may be properly considered one of the first specialists in laryngology in this country. His active work was done only a little over fifty years ago. This Association was founded in the year 1878 and embraced on the roll of its founders almost the entire number of those who were devoting themselves at that time to the specialty of laryngology in America. Consider the marvelous expansion since that day, both in the number of specialties and in the number of men, properly or improperly, announcing themselves as specialists in laryngology. The Seventeenth International Congress of Medicine, to be held in London this coming Auugst, announces twenty-three sections or departments in medicine. In place of the little group of twenty or thirty men in 1878, if we are to take the estimates given by competent authorities, there are in this country today alone, several thousand men described as laryngologists. In the state of New York a list has recently been compiled numbering several hundred.

---

*Read at the thirty-fifth annual meeting of the American Laryngological Association, Washington, D. C., May 7th, 1913.

A perusal of the early volumes of the Transactions of this Association will show the magnificent work that was done by the laryngologists of thirty-five years ago. The specialty, however, has extraordinarily developed, and today it means vastly more than it did at the time of the establishment of this Association. No longer is it confined to the treatment of diseases of the larynx. The closely associated and all important field of rhinology has been discovered, and demands equal or even more careful consideration; and while not actually a part, in practice otology, too, has become so wedded to laryngology that it is usually considered to belong to "greater laryngology." Bacteriology, with all that it means, has been born since the early days of laryngology, as well as the important field of bronchoscopy.

This rapid expansion and the increasing numbers of those who are announcing themselves as specialists raise a most serious question and one which is deserving the careful consideration of this Association. It is, what preparation or education is required in order to become properly equipped for the specialty? Already it has been the subject of an interesting discussion before the American Laryngological, Rhinological and Otological Society, and last year it was considered at the Liverpool meeting of the British Medical Association. As Shambaugh rightly points out, a proper training should give a man the ability to do not merely routine, but original work, something that we in this country have been sadly lacking in.

It cannot be emphasized too strongly that the clinical side is only one side of a specialty. It has been my privilege for many years to be engaged in postgraduate instruction, and I have had in this way an opportunity of meeting many men who are proposing to specialize. More and more I am impressed with the inadequacy of the preparation that the great majority make. The six weeks' specialist has been a constant reproach to postgraduate schools. This, however, is not altogether the fault of the school. The postgraduate school is only responding to the demand of the practitioner. If we are creditably informed, many a small town in the middle west possesses no less than five or ten "nose and throat specialists." Where they acquired their specialty is an unknown quantity. As Wishart, in a communication later to be referred to, very forcibly states, "Up to the present, on this continent, there is no recognized portal to the pursuit of the specialty. On the other hand, the gates may be said to be many, and yet, mirabile dictu, our towns and villages, and even our cities, are filled with "specialists" who have entered by no gate whatever, but have simply 'climbed over the wall,' and, to some extent at least, are to be considered merely as thieves and robbers."

The basis of all medicine is anatomy and pathology. This seems to be lost sight of by the man who too often appears to choose the specialty of laryngology because it is an easy way of practicing medicine and making money. Only recently in Vienna, we were told of an American who went there to get an opportunity to do a series of submucous resections, and who publicly asserted that that was all that he had come for, and that he had no wish or desire to acquaint himself with the anatomy or pathology of the subject. Only second in importance is a thorough training in general surgery. Our specialty has become to a large degree surgical, and we are called upon, as our predecessors never were, to perform all sorts of major operations. This demands a thorough grounding in every phase of surgery. Of general medicine and its profound bearing upon our specialty, it is unnecessary to speak.

Only after the young man has been thoroughly grounded, then, in anatomy, pathology, surgery, and medicine, is he prepared to take up the clinical side of laryngology. This preparation cannot be made in a month or a year. To our mind, it demands at least two years—and, better, three. We recognize, of course, the objection that will be raised in regard to the time required. This can be probably reduced to a considerable degree by a more thorough preparation in the undergraduate school, although the kind of training which we have in mind can never be entirely acquired by the student who is studying for a degree and who must prepare himself upon a number of subjects. What are the usual methods of preparation for special work at present? They are, first, learning the specialty in the course of a general practice, with or without connection with a special clinic. This is the method that many of the beginners in our specialty were compelled to pursue, and it continues to be the method pursued by many men who, in a natural way, graduate little by little out of general practice into some specialty. Usually the physician who is doing this is so occupied by the demands of his general practice that he can devote only a little time, and that with much irregularity, two or three times a week, to the clinic, gaining some instruction from his chief, but compelled to learn for the most part by himself. The objections to this method are manifest. It can never give a broad training, although it is bound to produce what is of inestimable value, a man well fitted by years of experience for special study.

The second method is by taking postgraduate courses here or abroad. There has been a marked advance in the method of instruction in our postgraduate schools in recent years. A student can get excellent short courses in every department of laryngology, as well as opportunity to work in the various clinics as long as he may desire. To our mind the value of the postgraduate medical schools cannot be overestimated. They have been and are doing a tremendous good for the physician. Their chief mission, in our judgment, however, is to the man in general practice who desires to brush up in order to do his work more efficiently. The four or six weeks' course should be entirely done away with, except for specialists who desire to do advanced work, and no beginner should be received for a period of less than six months, or, better still, a year, and then only after he has had the necessary groundwork in anatomy, pathology and surgery just referred to. This should take the form of connection with a laboratory and a surgical clinic, each for a period of at least a year.

Third, residence as interne or assistant in a special hospital. This offers, as far as the clinical side of the training is concerned, undoubtedly the best opportunities of acquiring the specialty. A young man who has gone through one of our special hospitals in this country or in Europe, residing there from one to two years, will undoubtedly come out well trained from the clinical standpoint. Such opportunities are, however, comparatively few, and even in those who have enjoyed them there is usually a sad lack of preparation in the groundwork to which we have just referred, and which is so essential for a thorough training.

That this question of education in medical specialties is receiving the consideration of the profession is evidenced by the action taken at the recent Clinical Congress of Surgeons held in New York last fall, in regard to the establishment of a postgraduate surgical degree. If this is true of the surgeon, is it not equally true of the man working in other departments of medicine? Dr. Wendell Reber, of Philadelphia, in a paper read before the Pennsylvania State Medical Association last year, on the teaching of ophthalmology in America, has advocated the giving of a degree in ophthalmology to men who have successfully carried out a required course of instruction. He believes that there should be some agreement between the fourteen postgraduate medical institutions in this country "as to the minimum training required of applicants for examination, and some approach to uniformity in the ordering of a three and a six months' course." We would, as we have said, favor a decidedly longer time than three months. "He recommends that no applicant for postgraduate teaching should be admitted to any of the well-recognized specialty courses who has not completed three years in general practice or its equivalent in general hospital work. If this idea should be carried out in connection with all the specialties in medicine, we would have in a few years a highly trained body of men to whom the public would turn as the men best qualified by education and experience to carry on their work." (Quoted from *The Medical Times.*)

To these views, in the main, we heartily subscribe. What we desire is quality in those entering our specialty, not quantity. The American method of getting results by the quickest means, together with the commercial spirit which too often obtains at the bottom, we must sadly confess has not escaped the noble profession of medicine. Specialism, with all that it represents for good, has unfortunately tended to offer an attractive field for commercial exploitation. It cannot be disputed for an instant that there are too many specialists. The public, it is our honest conviction, would, if two-thirds of them should be removed by marrying a rich wife, or some other equally certain but pleasant cause, be the better for their removal. The inauguration of some form of examination requisite to a degree would, beyond a doubt, work most beneficially in this direction.

We do not wish to give too pessimistic an opinion, but it is our experience, based on what we have seen here as well as abroad, that the man who has the proper idea of what specialism is, and what preparation for it is required, is a great exception. We can do no greater service to the specialty we love and represent than on every occasion to impress on those who have aspirations to specialize, the seriousness of the undertaking. Individual action alone, however, will not be sufficient. A concerted advance movement to properly standardize our specialty is essential. In our opinion, the time is ripe for such a movement. As a result of the discussion before the American Laryngological, Rhinological and Otological Society two years ago, a committee was appointed to consider the matter. This committee has spent much time in obtaining the views of American and Canadian laryngologists, which they have incorporated in a report which will be presented at the meeting of the Society this week. This report, prepared by Dr. D. J. Gibb Wishart, chairman of the committee, I have had the privilege of examining. It agrees in the main with the views expressed in this paper, namely, that a definite plan or scheme of instruction for special work should be determined upon. As an outline for a more definite working basis, this should consist of the following divisions: First, a thorough preparation in the undergraduate medical school; second, a general preliminary training through several years of practice or hospital interneship; third, at least six months of special instruction, covering not merely the clinical side, but the anatomic, pathologic, surgical, etc.; third, eighteen months of interneship in a special hospital or in the ear, nose and throat department of a general hospital; and fourth, at the conclusion, an examination by a university which should confer upon the successful candidate a postgraduate degree, such as Ph. D. (otolaryngology). The committee is strongly of the opinion that the postgraduate instruction should be given in some established university, and further, that to make the departure a complete success, legislation will be necessary. This, when public sentiment has been aroused, there will be no difficulty in securing. Finally, the committee is of the opinion that any success in this advance movement must be the result of concerted action on the part of the several national societies representing America and Canada, and this association will, in all probability, in due time receive a communication asking it to join in the further consideration of the proposed standardization.

XXVII.

CLINICAL INDICATIONS FOR THE MASTOID OPERATION.*

By W. Sohier Bryant, A. M., M. D.,

New York.

The history of the recent development in the diagnosis, prognosis, and surgical treatment in mastoid surgery is marked by four brilliant achievements: (1) The deductions from bacterial examination of the discharges; (2) the use of the Roentgen ray in disclosing intraosseous conditions; (3) the short convalescence, and (4) the improved hearing after the operation.

Formerly the accepted indications for mastoid operation were: Symptoms of cranial and hemic complications, labyrinthine suppuration, fistula of mastoid process, facial nerve paralysis, abducens nerve paralysis, fluctuation over mastoid region, edema over mastoid region, deep tenderness over mastoid region, rise of temperature, and headache, all associated with middle ear suppuration and chronicity.

These symptoms, in varying combinations, served as indications of the presence of mastoiditis and the necessity for opening the mastoid antrum. At times there was difficulty in estimating the true importance of the various symptoms. It often happened that cases which required immediate operation were delayed because of the absence of some of the classical symptoms, such as pain, tenderness, swelling and middle ear suppuration. Atypical cases, with absence of direct evidences of mastoiditis, were either not diagnosed until it was too late to save the patient's life, or a tedious recovery followed a delayed operation. Until comparatively recently mastoid operations were delayed on principle until softening of the bone had taken place. Expectant treatment held out the encouragement of convalescence without operation.

---

*Read before the Medical Association of the Greater City of New York, at the New York Academy of Medicine, February 17, 1913.

Formerly the prognosis and also the results of the mastoid operation were most discouraging. Usually the operation meant a slow convalescence with many painful postoperative dressings, and what was worse, considerable impairment, and sometimes complete loss, of all practical hearing. Small wonder is it that the operation was delayed as long as possible.

The present day indications for a mastoid operation include, in addition to the former indications, several definite elements of preeminent value in both diagnosis and prognosis. The most important of these indications are: First, rise of temperature otherwise unaccounted for and the toxic appearance of a patient suffering from inflammation of the middle ear or with a history of middle ear inflammation. Second, the infallible indication of the presence of streptococci mucosæ in the discharge, together with the determination of the morbidity of the mastoid, reinforced by the skiagram. Third, the demonstration of a solid mastoid in the presence of middle ear suppuration.

The histologic examination and count of the blood cells is useful as a confirmatory diagnostic and prognostic point. By itself it has little significance. When other symptoms are present, their value is so much greater that the deductions from the blood count can be disregarded without weakening the conclusion.

In the presence of streptococci mucosæ, or with the unaccountable rise of temperature and the toxic appearance of the patient suffering from middle ear inflammation, the operation for mastoiditis is infallibly indicated in cases where the skiagram shows a diseased or doubtful appearance. Either of these symptoms may be the only obvious clinical symptom. If there is any doubt that the case is one of mastoiditis demanding immediate operation, the skiagram will establish the final diagnosis.

Bacterial Examination.—Streptococcic infection of the mastoid is the most dangerous infection. Infection by staphylococci or pneumococci is much less likely to produce serious complications. Of the streptococcic infections, those patients infected with streptococcus mucosæ are apt to develop the most serious complications, both intracranial and hemic. An observation of the greatest importance is the fact that an infection of the mastoid due to streptococcus mucosæ runs a remarkably insidious course with absence of objective symptoms and with slight tendency to resolution without operation.

There are three striking facts about this bacterial differentiation, and these facts demand consideration. (1) The presence of these insidious bacteria often discloses cases requiring operation which would not be performed were it not for this demonstration. The infection is extremely virulent and at the same time the symptoms are latent. (2) Streptococcus mucosæ may cause little or no pain, and no swelling. (3) The presence of this organism is usually indicative of extensive bone involvement or complications, so that it becomes in itself one positive symptom.

Involvement of the mastoid by streptococcus mucosæ demands an immediate operation. If, in spite of this symptom, the operation is delayed, it will be found that soon an emergency operation is demanded.

Skiagram.—The skiagram made by the Roentgen rays is another source of evidence most useful in determining a diagnosis in infections of the mastoid. Like the bacterial differentiation, the skiagram is of especial value in cases which, without its use, might be overlooked. In most cases the skiagram is merely confirmatory; in doubtful cases it gives the prognosis and determines the operation.

The skiagram should accurately portray the external topography and interior structure of the mastoid. It can show empyema of pneumatic cells, softening and abscess formation in the mastoid and surrounding parts, as well as the position of the antrum and sigmoid sinus. The accuracy of the deductions is of course enhanced by the comparison with the sound side, in spite of the fact that in a large number of skulls the internal structure of the mastoid has been found to differ markedly on the two sides.

It has been observed that in normal temporal bones the interior structure of the mastoid varies considerably, grading from pneumatic cellular bone through diploetic bone to dense ivory bone. Clinical observation shows that mastoid infection in the pneumatic mastoid tends either to formation of frank mastoid abscess or to resolution, whereas in the mastoid where diploetic or dense bone surrounds the mastoid antrum, a mastoid infection does not form a frank mastoid abscess, nor does it tend to resolution. It tends rather to eventuation in chronic middle ear suppuration or to the formation of the serious intracranial and hemic complications.

The shadows of the skiagram show opacities, the outline of the bone, the internal structure of the bone, and the position of the sigmoid sinus in relation to the antrum and canal. From clearly outlined cells we find varying degrees of opacity to the condition where the whole region is entirely opaque. This total opacity is due either to dense bone or to pus. The intermediate degrees are due to relative degrees of inflammation or thickening of the bone. These conditions are differentiated in this way: As long as the boundaries of the cells are distinct, one infers that there is absence of inflammation. Blurring of the outlines indicates inflammation.

The skiagram also gives a differential diagnosis between middle ear suppuration complicated by otitis externa without mastoiditis and otitis externa with mastoiditis.

In cases where both ears are affected, the skiagram is useful in determining which ear demands operation or whether both ears should be operated upon.

The skiagram shows abnormal bone edges, demonstrating erosions of the bone due to granulations, cholesteatoma and new growths.

The skiagram shows the presence of epidural abscess and perisinus abscess. In fact, the skiagram gives so much important information that the Roentgen ray examination should be employed in every case of middle ear suppuration and in cases of suspected latent inflammation.

A point to be emphasized in connection with the employment of the Roentgen rays is the fact that the skiagram should be made by an expert and should be interpreted by the same expert.

When shall we operate? The means by which we determine the time for operation are: (1) The history of the case; (2) the condition of the ear, as determined by inspection; (3) the presence of general sepsis; (4) symptoms indicating intracranial complications; (5) the formation of an abscess which requires surgical drainage for its proper treatment; (6) a skiagram of the mastoid region; (7) the bacterial examination; (8) chronic middle ear suppuration which will not yield to other treatment and with cholesteatoma; (9) acute suppuration of the middle ear which has become unreasonably prolonged; (10) acute suppuration when the skiagram shows an absence of the antral pneumatic cell system, and (11) suppurating labyrinthitis. Recourse to the Roentgen ray clears up a doubtful diagnosis, giving, in connection with the other clinical symptoms, positive indications for or against the mastoid operation.

In a dense mastoid process the suppuration does not tend to resolution but to continuation and to the development of the dangerous complications of mastoiditis. In a pneumatic cellular mastoid, inflammation goes on either to abscess formation or to resolution. In mastoids partly dense and partly pneumatic, with the dense areas occurring near the antrum, the behavior of infection is identical with the wholly dense mastoids. These observations show us that with inflammation of the mastoid shown by discharge, skiagram, etc., a mastoid operation is indicated in all but the pneumatic bones, and in them when mastoid abscess has been formed. In other words, in the pneumatic mastoid suppuration, delay the operation until the formation of abscess; while in the dense mastoid, suppurative inflammation of the mastoid is sufficient indication for mastoid operation. In these cases, abscess formation should not be waited for.

The prognosis following the mastoid operation, if the operation is delayed, is much the same as formerly, but the cases which have been operated on at the earliest crucial moment when operation is indicated give a prognosis of speedy convalescence, absence of complications and restoration of normal hearing.

We now know that delaying operation after the crucial time has arrived for an operation results in prolonged convalescence, impaired hearing, intracranial and hemic complications, and their serious consequences.

Prolonged suppuration of the middle ear is a danger to the patient and also progressively impairs the hearing. Since this condition is usually dependent on inflammation of the mastoid antrum, a mastoid operation is demanded both for the welfare of the patient and in order to preserve as much of the hearing as possible.

In conclusion, I would say: (1) Operate when mastoid abscess is present; (2) operate on mastoiditis due to streptococcus mucosæ; (3) operate in the presence of intracranial or hemic complication of middle ear infection; (4) operate on mastoiditis, acute or chronic, when the bone is of the solid variety.

The former indications for mastoid operations were, as we know, very vague. With our more definite and more specialized indications, there is no reason to delay an operation which, in the end, must be performed. An early operation shortens the length of the disease and also shortens the length of convalescence. In other words, there is a quick convalescence from the disease itself, and there is a quick convalescence of the patient, because the course of the disease has been curtailed.

In the meantime, please note that the possibility of serious complications has been prevented.

It is impossible to lay too much emphasis on the fact that we should determine as early as possible whether an operation is demanded. If an operation is demanded, operate at once, and do not wait until the hearing mechanism is so impaired that it will be impossible to restore it. The sooner the operation, the less destruction of parts and, therefore, the less impairment of the function.

XXVIII.

CONGENITAL OSSEOUS OBSTRUCTION OF THE POSTNASAL ORIFICES.*

By C. W. Richardson, M. D.,

Washington City.

In July, 1912, there came under my observation a case of complete osseous obstruction of the postnasal orifices at as early a period in the life history of the case as any other observer has seen such a condition, and at a much earlier period of life than have the majority of the reported cases come under competent medical observation. In an investigation of the literature on the subject, I find only a few cases in which the obstruction was recognized in infancy.

According to Dr. R. Kayser, in Paul Heyman's Handbuch, the first case reported and operated upon in the living was made by Emmert in 1854. Otto, in 1830, reported the first case of closure of the choana in a fetus, and next follows the interesting case reported by Luschka in a newborn dead child, 1859. Following these reports we find numerous cases of the condition reported from the anatomic table, the newborn infant and in child and adult life. It has not been possible for me to tabulate all the recorded cases in the literature, but I should judge that they do not exceed one hundred in number. It is interesting to observe that a fair proportion of cases has been reported by members of this association. The obstruction of the postnasal orifices may be membranous or osseous.

The membranous obstructions are usually formed posterior to the nasal cavities in the nasopharyngeal cavity, but lay in contact with the postnasal orifices so as to completely obstruct them. They extend from the upper surface of the soft palate at the union with the hard palate, and extend to the roof of the postnasal cavity.

The osseous obstructions are usually placed within the nasal chamber within a millimeter or more from the free border of the posterior nasal orifices. They are usually composed of thin plates of bone, and may be unilateral or bilateral. To the two above mentioned forms may be added the congenital atresias by which the bones entering into the formation of the postnasal orifice become united, thus more or less completely obstructing the postnasal orifices. Various forms of atresias, excited by disease and deformity, cause obstruction to the choanæ, but, as these are not congenital, they do not enter into the scope of this paper.

---

*Read before the meeting of the American Laryngological Association in Washington, D. C., May, 1913.

On July 30, 1912, I was urgently requested to visit in consultation, early in the morning, an infant just born, who had marked difficulty in breathing. Dr. Wynkoop, who accompanied me on my visit to the infant's residence, told me of the nature of the case and the probable seat of the difficulty. When the infant was brought to me I was impressed with the peculiar cycle of respiratory difficulty with which it was contending. It would lie at rest for barely a moment, with its lips drawn tightly and its cheeks slightly indrawn, when it would begin to struggle for air, the face becoming suffused and slighty cyanosed, the condition being relieved as the child began to cry. As soon as its discomfort was relieved through the mouth breathing necessarily instituted while it was crying, it would cease to cry, with a recurrence of the difficult breathing. The cycle was immediately suspended when the lower lip was pressed down so that uninterrupted mouth breathing was permitted. Examination of the mouth and fauces demonstrated that all the structures in these parts were in normal relation and anatomically perfect. Examination of the nasal organ demonstrated that it was normal in external appearance. Inspection of the nares anteriorly, after removing the copious secretion, evidenced these to be in a normal condition —no adhesions or new growths. Washing out each cavity separately demonstrated that no fluid entered the pharynx, and, after the cavity became filled with fluid, it returned in excess by the anterior nares. The use of a probe proved that at a depth of about five centimeetrs the further progress of the probe was arrested by coming in contact with a firm, rigid, resisting wall. The sensation imparted through the probe when introduced to the bottom of the nasal chamber was as though the end of the probe was impinging upon a firm, resisting plate of bone. The sound conveyed by the impact of the probe was similar to that imparted under similar conditions when a probe strikes a thin plate of bone thinly covered with soft tissues. It was impracticable, as well as considered unnecessary, to make an attempt to examine the postnasal cavity through digital exploration, as the character of the respiratory difficulty and the examination through the anterior nares demonstrated, without doubt, the deformity existing must be a complete obstruction of the postnasal orifices. The maintenance of respiration until voluntary mouth breathing was established and the maintenance of nutrition presented problems to be considered in the management of this case. It seemed that an attempt at operative relief was only to be considered if the life of the child became endangered through its inability to maintain mouth breathing or through malnutrition. The operation itself, without proper protection in the nasopharynx, might be attended with immediate danger, while the hemorrhage and resulting reaction were factors to be considered in an infant so young. Even should the operation become imperative and the procedure have been successfully accomplished, the problem of preventing the closure of the artificially made opening in one so young would have indeed been very difficult. Through a relay of assistants the child's lips were kept constantly apart so that continuous mouth breathing could be maintained, and, through this assistance, at the end of its second week of life, it had learned to voluntarily maintain mouth breathing, thus overcoming one of its great life problems. Its nutrition was maintained very satisfactorily by feeding it through the aid of a "dropper," thus solving the other problem. In a short period after establishing voluntary mouth breathing she learned to manipulate the bottle, alternating in sucking and breathing through the buccal cavity. From this time on the child has continued to develop as any child with normal respiratory organs.

The question to be considered in a congenital occlusion of the choanæ, especially of the osseous type, is the proper time for operative interference. The danger attendant and the difficulty of operating upon one so young, with the impossibility of maintaining the patulency of the opening, after the obstruction has been removed, would seem to argue against an immediate operation if it be possible to maintain breathing and nutrition. The marked success obtained in the case that I present to you would seem to favor the expectant surgical policy in such cases, as well as the successful issue in many other cases. It would seem that the proper period for such cases would be as early as surgical interference could be done with perfect safety and with an absolute assurance that the manipulation could be made through a cavity large enough not to be embarrassing for want of size, and with a postnasal space large enough to permit of easy introduction of the finger to note the obstruction from behind, as well as to afford protection and serve as a guide. The operation should nevertheless be done as early in life as possible, when the above mentioned conditions have materialized, so that any alteration of the facial skeleton may be forestalled and before any pathologic process has been started in any of the neighboring tissues or organs. It seems to me, therefore, that nothing having arisen to disturb the normal progress of a child with congenital occlusion of the choanæ, about the second year of life would be the most appropriate time to undertake surgical interference.

XXIX.

INTRAOCULAR TENSION IN GLAUCOMA LOWERED BY INJECTION OF THE SPHENOPALATINE GANGLION.*

By H. Edward Miller, M. D.,

St. Louis.

I wish to record an observation which, as it stands, is simple and does not exhibit any brilliant result, although it proves beyond question an influence between the sphenopalatine ganglion and intraocular tension in glaucoma.

In the December, 1908, issue of the *American Journal of Ophthalmology,* Dr. A. E. Ewing called attention to the relief of pain afforded in glaucoma by cocainizing the sphenopalatine ganglion.

No specific influence has heretofore been proven to exist between the tension and medication of the ganglion.

I am indebted to Dr. W. H. Luedde for the opportunity and cooperation in this case, to which he referred among others, in a paper, "The Usefulness of Schiötz Tonometer," at the meeting of the Medical Association of the Southwest, held at Hot Springs, Ark., on October 9, 1912.

August 24, 1912, Mrs. T. T., a white patient, aged 55 years, had a simple chronic glaucoma involving both eyes. She had been thus afflicted for the past ten years. Five years ago, while a patient at the O'Fallon Dispensary, the sight in her left eye was fair, but her faculty of vision in the right no longer enabled her to read. Both visual fields were much contracted. The use of myotics apparently controlled the condition. The patient refused any operative interference and then disappeared for several years. When she again came under observation, her right eye was absolutely sightless, not even the faintest light perception remaining. The sight of the left eye was so much reduced that she could not find her way about. During the absence of over three years she had been without any treatment except during a brief stay at the Johns Hopkins Hospital of Baltimore, where she again refused to be operated.

---

*Read before the Oto-Laryngological Section of the St. Louis Medical Society, March 26, 1913.

The use of myotics had been entirely stopped. Upon resuming them now, Schiötz tonometer registered a prompt reduction in the tension in both eyes, the right from 50 mm. to 30 mm., and the left from 36 mm. to 18 mm. Three days later she came to the office complaining of intense pain in the head, which she ascribed to "catching a cold" while sitting in a draught the night previous.

She had never experienced any pain before. Tonometer examination showed the tension had risen in each eye to a higher point than at any previous measurement, viz., right eye 60 mm. and left eye 51 mm. The sight in the left eye, which had been much clearer after the use of myotics, was "cloudy" again.

Without any ocular treatment the patient was brought to me for a nasal examination, which disclosed a severe general pansinusitis. The swollen membrane was shrunk, and cocain was applied to the area covering the sphenopalatine foramina, with the result that Schiötz tonometer within three-quarters of an hour registered a drop from 60 mm. on the right to 55 mm., and on the left from 51 mm. to 37 mm., before any myotic was used.

Under the combined use of myotics and the nasal treatments the tension became less and less during the next three days, reaching 32 mm. in the right eye and 26 mm. in the left (a drop of 28 mm. in the one and 25 mm. in the other).

The patient was now compelled to return to her home, some distance in the country, but subsequently reported much improvement; headache had almost entirely disappeared and her sight was clearer. The first few days an attendant accompanied her, but now she was able to get about the city alone.

December 24, 1912, patient returned to the city with both eyes in "bad condition for the past three weeks." Treatments at home had been very irregular and unsatisfactory. The tonometer registered 70 mm. in each eye, a rise of over 40 mm. during her absence, and higher than at any time during previous observation. The sight in the left eye had fallen from the ability to read letters about one and one-half inches long at five feet, to distinguishing fingers at one foot.

The marked relief afforded by the combined treatment of her nose and eyes before, brought her back with the request that the same procedure be resumed.

The nasal tissue was too greatly swollen to permit of accurate application to the ganglionic area, but no sinusitis existed at this time. Myotics again reduced the tension, especially in the left eye, and sight became clear enough to see large letters at four feet on the third day.

Two days later a subconjunctival injection of 5 per cent sodium citrate solution was made in the right eye, resulting in a markedly lessened tension in both eyes, even greater in the eye opposite the one injected, dropping 20 mm. However, within two days it was as high as before. Nasal treatments were continued, but the futile attempt to make an appreciable impression upon the tension induced me on January 13, 1913, to begin the ganglion injection method as described by Dr. Greenfield Sluder, December 30, 1911, in the *American Journal of Medical Sciences*.

I used a straight needle, and estimated about one-third cm. penetration in a direction backward, upward, and outward from the bony posterior tip of the middle turbinate. About six minims of a 5 per cent alcoholic solution of phenol was injected. Patient immediately felt a "new pain" starting in back of her ear and extending down her neck to the shoulder. This disappeared in about two hours. Tension quickly rose, but on the following day had dropped fully 20 mm. Directly after injection the patient complained of complete loss of sight, but this soon disappeared, and she could see better than at any time during the past four months. On the morning of the fourth day after this first injection a rise of about twenty points was noticed, and now the right ganglion was injected. Myotics had been regularly continued all this time. The same gratifying bilateral drop in tension was produced and a greater clearness of sight in the left eye.

Failure to obtain the typical reaction after injection ofttimes results from the inability to reach the ganglion.

A total of seven injections were made, of which two should be dismissed; in the one the needle failed to reach the ganglion and likely entered the sphenopalatine artery, for a profuse hemorrhage occurred and the patient weakened so as to become bedridden for several days; the other injection followed the news of severe illness of her husband and the request for the patient to return home against her wishes.

Unexpected excitement always caused quite a rise in tension in this case. Irritants, such as formalin or argentum, had no apparent effect on the tension when applied over the sphenopalatine foramen.

Averaging the other five injections, we find the immediate result after injection was a rise of an average of 10 mm. Within twenty-four hours the average drop was 17½ mm. from the original scale. The minimum drop was 7 mm. The maximum was 28 mm.

XXX.

PERITONSILLAR PHLEGMON—REPORT OF A CASE.*

By F. E. Hopkins, M. D.,

Springfield, Mass.

Limited by the character of the case I have to report, my title should be phlegmon of the peritonsillar space, rather than the comprehensive one of "Phlegmon of the Upper Air Passages," under which it appears in the program. The principal point which it illustrates is the possibility of erosion of large blood vessels.

As to the cause of peritonsillar infections, we readily accept the bacteriologist's dictum that the throat at all times harbors bacteria ready marshaled for invasion; and since the crypts of lymphoid tissue are portals easily assailed, infections of this region should be frequent and systemic involvement severe. The wonder is, not that phlegmons threatening life occasionally develop, but rather that such serious invasion is so rare that an active professional life may pass and not a single case come under observation.

I leave to the bacteriologist the naming of the invading host and of the allies which join with eager savagery in the campaign; and I leave to him, too, the naming of the armies of defense, both of the first and of the second line, adopting for myself the general terms of virulent or aggressive infection, and a lowered resistance on the part of the patient. Pardon this seeming levity—it is only seeming. Bacteriologic literature, with its explanations of infection and resistance, gives this kind of a picture, and though the language of this branch of science is not at my command, the reference will be understood.

Such microscopic examinations of pharyngeal phlegmon as I have on record give as a usual finding staphylococcus aureus, with in some cases a mixed infection—the most troublesome addition being streptococci. The staphylococci alone fulfill their contract for destruction with less systemic disturbance than when working in alliance with others.

---

*Read before the American Laryngological Association, May, 1913.

However, microscopic findings are of little value in determining the treatment of these cases. The stage of progress decides that, and early, effective drainage is the best assurance of a favorable prognosis.

Peritonsillar phlegmon may run its course to serious complications with less systemic disturbance—as shown by prostration, temperature curve and pulse rate—than is the rule in follicular tonsillitis. The pain and distress may be great, with the temperature not above 101°, and a pulse of from 80 to 90.

Suffocation from flooding the larynx by the sudden rupture of the abscess has been reported, and tracheotomy has been required because of closure of the phraynx by infiltration and edema; but the complication I wish to emphasize is that of erosion of blood vessels by the necrotic process.

When only the tonsillar artery is involved the hemorrhage may cease spontaneously or be easily controlled; if the ascending pharyngeal, the situation is very serious, while a break in the wall of the internal carotid brings a swiftly fatal end. As illustrating invasion of the first or smallest of the three arteries, Dr. Bates of Springfield has recently described to me a case of active hemorrhage following the spontaneous rupture of peritonsillar abscess. Bleeding ceased without a resort to either pressure or ligation.

Bosworth's work, with its painstaking research of literature, was published twenty years ago, yet the chapter on peritonsillar phlegmon is still the fruitful source of reference. He gives a list of twenty authors, who, over a period of forty years, had reported cases of involvement of the large vessels during the course of phlegmon. Of all the cases reported but five recovered, and the inference is that in these only the smaller vessels were eroded.

Nearly all the recent textbooks speak of the possibility of this destructive invasion of blood vessels, but none of the authors I was able to consult mentions a case or refers to one.

The case whose brief history follows was under the care of Surgeon Major E. A. Gates of Springfield, Mass. I saw the patient in consultation on May 23, 1900, and found: H. M., male, American, twenty-six years of age, of poor resistance because of irregular life and recent acute illness. He had suffered from measles early in the month, and while convalescing had taken cold—according to his own statement —the particular manifestation of this cold being a sore throat. There was a marked peritonsillar swelling upon the left side, with severe pain, but the patient was not prostrated, being at this time dressed and about the house. His pulse was 80 and temperature 100,° and during most of this illness the pulse rose only to 80 or 90, and the temperature at no time exceeded 101.° No fluctuation could be made out. I saw him again on the 24th, and although the cellular infiltration had increased there was no evidence of "pointing," nor could fluctuation be felt. Deep incisions were made, but these yielded no pus, and little bleeding took place. Two days later a hemorrhage occurred, which the attending physician controlled by pressure. A terrific hemorrhage recurred on the 27th and proved quickly fatal. A postmortem was not allowed, but such examination as could be made immediately following death showed rupture through the posterior pillar —the flood from the eroded carotid finding exit here.

Such a mass of cellular infiltration should be explored by a blunt instrument, even the finger, following unfruitful incision. In this case, the patient—an only and spoiled child—objected vigorously, as did the parents; and as a peritonsillar abscess usually ruptures into the incision or burrows its way to the surface, further interference was not insisted upon, with the unusual result named.

XXXI.

PRIMARY TUBERCULOSIS OF THE MIDDLE EAR.

By Chas. H. Long, M. D.,

Chicago.

There is probably no disease to-day more widely discussed, not only by the physician but by the laity as well, than the "Great White Plague." For many years it has claimed the attention of some of our greatest scientists. Koch's discovery of the tubercle bacillus in 1882 was of unprecedented value to the profession; and since that time, particularly during the last decade, marked advancement has been made in the knowledge of tuberculosis. While clinical research continues, efforts are exerted to utilize each progressive step for the enlightenment and benefit of mankind. While some theories have been so conclusively proved by experimentation and application that they have become of universal practical value in diagnosing cases and for the treatment and prevention of tubercular infection, there are still many questions open to discussion. Concerning these, we must accept the opinion of the majority until more extensive investigation and time shall prove or disprove them.

Before proceeding to the subject of primary tubercular infection of the middle ear, which is the purpose of this article, let us take a brief survey of the present tubercular situation as it is presented to us by recent scientific workers and by personal study and observation.

There has been much disputation as to whether the human and bovine tubercle bacilli differ or are identical. Theobald Smith claimed to have found "many distinct differences." Koch[1] believed the difference to be one of type, rather than species, the position now taken by all who support a differentiation between the two groups. Koch further stated his belief that this difference is sufficient to influence the combating of the disease. Others[2] believe in the unity of the human and bovine bacilli, maintaining that, while there are marked differences in cultural growth and virulence between the two "so-called types," these types vary within themselves in the same way to such an extent that the difference in the groups overlaps, making it impossible to consider the one group as an entity distinct from the other. The English Royal Commission[3] confirms this instability of type, and concludes that the gap between the two types is not a wide one, while Whitla[4] states that the conclusion of the "great majority of recent workers is that the human and bovine types of bacilli are identical."

Whatever the decision in this regard, there is now a general agreement upon one point,—the danger to man of infection from both types of bacilli. The English Royal Commission, in their report of 1911, state as follows: "We have investigated many instances of fatal tuberculosis in the human subject in which the disease was undoubtedly caused by a bacillus of the bovine type, and nothing else. We have compared the lesions from such cases with those obtained from parallel cases of fatal tuberculosis in which the human tubercle bacillus alone was discovered. Except for the difference in the type of bacillus found in them, these two groups of cases presented similar features; the clinical histories of the cases were alike, the cases all terminated fatally, and the lesions examined after death were found to be anatomically indistinguishable." To quote Professor Ravenel:[5] "Laboratory investigations have shown beyond question that bovine tuberculosis is transmissible to human beings and that the lesions produced do not differ in any way from those caused by human bacilli."

Considering these facts, is it then worth while to pause to discuss the much agitated question of the possibility of the human bacillus affecting cattle? It is interesting and perhaps valuable to know that the resistance of bovines is so great that the feebly virulent human bacilli can ordinarily have but little effect upon bovine tissue. This, however, seems not the salient point in the matter. When we observe the destructive inroads made by the bovine bacilli in the human being, does not the question of bovine susceptibility shrink into insignificance?

Again, in view of these facts, whatever its source, water, milk, milk products, or ordinary dirt, the infection of mankind by bovine tubercle bacilli should receive our careful attention.

Though the extent of danger is still in doubt, if we adhere to von Behring's[6] theory of latent infection, if we incline toward a growing conviction that, after all, the one type may be transformed into the other after long-continued residence in a particular host with all conditions requisite and favorable to such transformation, or if we advocate the popular theory[7] that the surgical forms of tuberculosis are due to bovine bacilli, which may also produce miliary tuberculosis; in any case, we cannot but be convinced of the perils of bovine bacilli to mankind. We do not, as some accuse, "minimize" the danger of infection by human bacilli by emphasizing the work of the bovine type. We merely hold that the latter infection has a significance which may not be generally appreciated.

It is universally recognized that age is an important factor in the infection of tuberculosis. The disease is considered preeminently one of childhood. The physical conditions, environments, and food of the child render it practically impossible for children to escape the inception and harboring of the bacillus. Though its presence is not always immediately evident, the seed is sown and awaits only the opportune moment, favorable soil conditions and lowered resistance to begin its destructive activity. The prevention of implantation is one of the means of eradicating the tubercular evil. Then must we consider first the question of tuberculosis and the susceptible child. Though much is being attempted to better existing conditions, yet more should be done. We fail to see how civic governments can consider it unnecessary to improve the public milk supply, in the face of such statements as the following, made by the English Royal Commission:[8] "A very considerable amount of disease and loss of life, especially among the young, must be attributed to the consumption of cow's milk containing tubercle bacilli."

It has been fully demonstrated that certain tissues of the human body exhibit a greater susceptibility for the tubercle bacilli than others. The lungs offer the best example of these, and, in consequence, are most frequently primarily infected. On the other hand, some tissues are so rarely invaded that many believe that their infection as a primary condition never occurs. To this latter class belongs the middle ear. Although Milligan,[9] after years of observation, concludes that primary tubercular infection of this organ is a comparatively common condition, he meets with little support and much contradiction of opinion; some experienced otologists even maintaining that the infection is never primary, being always secondary to the disease elsewhere, usually with a pulmonary focus. While few assume this extreme position, yet the general consensus of opinion inclines toward the rarity of the condition, which is not surprising when we consider the limited number of cases reported and the great difficulties in eliminating early, or latent tubercular infection.

Until the year 1903, according to Goldstein,[10] but three cases of primary infection of the ear had been reported. To these he added four. Recently, others have been presented, and, although there may be doubt as to some of these being primary, the remaining unquestionable ones give sufficient proof of the existence of a primary infection of the middle ear. Until it can be proved that some one part of the human system is immune to the invasion of the tubercle bacillus, the possibility of this condition cannot be authoritatively contradicted.

The following case which I have to report, appeals to me as one of special interest, in view of the extreme youth of the patient.

Case 1.—G. W. English, age, eleven months, female. According to the parents, the child at the age of one month appeared to have a severe cold. Both nose and eyes discharged for a couple of days, which was followed by a discharge from the left ear. The family physician treated the ear for some time by syringing, but no improvement resulted. February 1, 1910, she was brought to my clinic at the Post-Graduate Hospital by the mother. Examination revealed a profuse fetid purulent discharge from the left ear. The lining of the external auditory meatus was red and irritated. The tympanic membrane was absent. The probe revealed necrotic bone. The examination of the throat showed enlargement of the pharyngeal tonsil, which seemed to interfere with nasal breathing. This was removed. In all other respects the child appeared perfectly healthy. The treatment applied consisted of ordinary cleansing and Politzerization until April. No improvement resulting, a smear was taken and submitted to Professor Zeit of the Northwestern University for examination. He reported the presence of tubercle bacilli in the discharge. Owing to the child's youth, it was impossible to determine the exact condition of the hearing, but it appeared little impaired. The cervical glands were not enlarged and there was no tenderness or swelling over the mastoid process.

April 29, 1910.—The radical mastoid operation was performed. Only remnants of the malleus and incus remained. A mass of granulation tissue virtually buried the stapes, the granulations extending into the antrum. All were carefully curetted away with the exception of those around the stapes. The postauricular wound was dressed open. Owing to the rapid growth of immense flabby granulations, healing was protracted and many currettements were necessary.

June 2, 1910.—The postauricular wound had healed and the meatus was dry. About the middle of May the right ear was found to be discharging. Professor Zeit reported a pneumococci infection, but no tubercle bacilli. This ear was soon well.

October, 1910.—The child was suffering from a cold, and both ears were discharging after being dry for four months. No tubercle bacilli were discovered, and the response was negative to both the Moro and von Pirquet tuberculin tests.

October, 1911.—The last report of the case came at this time. Both ears were dry and, according to the mother, the child was in general good health. Up to the time I first saw her this child had never been given cow's milk or artificial food of any description. She did not appear to suffer pain in her ear, while she was so noticeably good natured that callers were impelled to remark about the attribute.

In our conclusion that this was a simple case of primary tuberculosis of the middle ear, we considered the following: The child's youth at the period of onset of the disease, its continuous general good health, the absence of tubercular infection elsewhere, as proved by the negative tuberculin tests after the radical mastoid operation, and the ultimate satisfactory condition of the ear.

As to the source of the disease, little need be said, although the child, to the best of our knowledge, was given no cow's milk or artificial food, the ignorance and carelessness of the laity afford ample opportunities for infection. The theory of predisposition and inherited tendencies was not regarded here, as there had been no instance of tuberculosis in the family and the parents of the child were in perfect health. The case being a surgical one, suggests a bacillus of the bovine type and the attack a result of dirt infection.

While there are many possible avenues for the inception of the disease, the features of the case eliminated all but two—the eustachian tube and the external auditory canal, with the probabilities in favor of the former.

The examination of the ear discharges determined the nature and localization of the disease, and the radical operation was performed in time to arrest its destructiveness. In support of this operation, which is often criticized, we maintain that if any operation is advisable, it should be of a radical nature, especially when the disease is localized. Surgeons have regretted its omission when further developments showed that its timely performance might have prevented a fatal termination of the disease.

Despite the constant advice to be ever upon the alert for the first indications of the tubercular disease, some of us fail to recognize the infection in its incipient stage. If the physician and bacteriologist would more frequently cooperate, there would be fewer disappointing results. While this reference is but an added stroke to the well worn nail, its insertion may be of benefit to some co-worker.

Let us not slumber while the foe invades; then become pessimistic, but "take arms against a sea of troubles, and by opposing, end them."

REFERENCES.

1. Koch, Robert: International Congress on Tuberculosis. British Medical Journal, October 17, 1908, Vol. II, p. 1190.
2. Arloing: International Congress on Tuberculosis. British Medical Journal, October 17, 1908, Vol. II, p. 1190.
3. English Royal Commission: Final Report on Tuberculosis. British Medical Journal, July 15, 1911, Vol. II, p. 122.
4. Whitla, Sir William: The Etiology of Pulmonary Tuberculosis. British Medical Journal, July 11, 1908, Vol. II, p. 63.
5. Ravenel, Mazyck P.: Transmission of Bovine Tuberculosis to Human Beings. Journal American Medical Association, December 18, 1909, Vol. 53, p. 2115.
6. Behring, E. von: "Ueber Lungenschwindsuchtentstehung und Tuberkulosebekämpfung. Deutsche med. Wochenschrift, Vol. 29, p. 689.
7. Raw, Nathan: Human and Bovine Tuberculosis. British Medical Journal, 1908, Vol. II, p. 1088.
8. English Royal Commission: On Human and Animal Tuberculosis. Second Interim Report. British Medical Journal, February 9, 1907, Vol. I, p. 326.
9. Milligan, William: Transactions Sixth International Otological Congress, 1900, p. 34.
10. Goldstein, M. A.: Primary Tuberculosis of the Ear Followed by Mastoiditis. Reported four cases. Journal of Laryngology, Rhinology and Otology, March, 1903, Vol. 18, p. 113.

XXXII.

REPORT OF A CASE OF TEMPOROSPHENOIDAL ABSCESS OF OTITIC ORIGIN—OPERATION—RECOVERY.*

BY JAMES F. McCAW, M. D.,

WATERTOWN.

The subject of this report was a female, 44 years of age, married, seen in consultation on April 27, 1912, at the City Hospital. The following history was obtained: She had suffered from a severe head cold six weeks before, which was followed in a few days by severe pain in the right ear and discharge thirty-six hours later, with only silght relief of the pain; these symptoms continued, and for about three weeks before coming to the hospital, she had suffered such severe pain over entire right side of head that the physician in attendance had kept her under the influence of large doses of morphin and free administration of whisky. On the above date another physician was called, and realizing the gravity of her symptoms, sent her to the hospital at once and asked me to see the case.

Upon examination her temperature was 100.6° F., pulse 102, respirations 20. Her face showed great suffering and anxiety; there was a very profuse, thick, creamy, purulent discharge from the right auditory canal, the membrana tympani was intensely congested, thickened and edematous; there was a small oval perforation in the lower posterior quadrant, through which pulsating pus was being discharged. There was such marked bulging and drooping of the upper posterior quadrant and the posterosuperior canal wall that the perforation was just visible. There was decided swelling and edema over the entire mastoid area and extending well above the antral region. This entire area was exquisitely sensitive to touch and severe pain elicited from the slightest manipulation. On assuming the erect posture in bed, dizziness was complained of. Mentality seemed dulled, but all questions were answered correctly; no nausea or vomiting. Convulsive seizures were absent, and no evidence of muscular paralysis. Both eye grounds were normal. Urine analysis negative.

---

*Read before the American Otological Society at Washington, D. C., May 6, 1913.

I operated upon this case the next morning, using the usual mastoid incision. After elevating the flaps and periosteum over the mastoid area and also from the tip, a very extensive bone necrosis was encountered, so that the lower half of the outer table, including the tip, was a sequestrum and was lifted out whole. From necrosis of the inner table, about an inch and a half of the sigmoid sinus was exposed, covered with pus and exuberant granulations. After cleaning out all necrosis and granulations, my attention was next given to the tegmen antri, which was soft, and with a curette and rongeur this was carefully cut away, exposing the underlying dura, which was very much thickened, discolored and felt resistant. An incision through the dura at this point opened directly into an abscess cavity from which was evacuated about a dram and a half of pus. The cavity, which was about one and one-half inches in depth, was carefully wiped out with cotton sponges, and upon examination there was no apparent limiting membrane, but the cavity seemed to be directly within the brain tissue, clearly demonstrating that it was a very acute and recent process. The outer opening through the dura was enlarged and a strip of iodoform gauze was carried in and packed loosely into the cavity. The mastoid wound was dressed in the usual way. Blood count at this time showed a leucocytosis of 13,200 and a polynucleosis of 84 per cent.

For three days following the operation the patient complained of considerable and severe pain over the right side of head, with nausea and vomiting, restlessness and irrational periods. The temperature kept steady between 100° F. and 100.8° F. during this time, and the pulse from 78 to 90. Blood count, taken each day, showed diminishing absorption with good resistance, so we began to look about for something else to account for this train of untoward symptoms, which seemed quite alarming, when it occurred to the writer that perhaps the withdrawal of the amount of whiskey and morphin which she had been accustomed to for six weeks was responsible for this postoperative development. With this idea she was put on chloral gr. iii and sodium bromid grs. xv, three times daily and at night if necessary. It was a most noticeable fact that all the cerebral symptoms disappeared promptly, her temperature was normal two days later, and she had no further symptoms of this character.

The first dressing was done on the fifth day, and the gauze removed from the abscess cavity, which was perfectly clean and without retention, the dural opening patulous. The cavity was dried out with pledgets of sterile cotton and one strip of iodoform gauze inserted. It was dressed every second day, and on the ninth day following the operation all gauze was left out of the abscess cavity, as it had contracted down to almost nothing and simply showed a depressed granulating surface. From this time on the patient made an uneventful recovery.

This report is made not so much for its importance as a contribution to this society, but simply that it may be on record for its clinical value. However, there are several points of interest in it from a clinical standpoint. The first to be noted is the difficulty in making a correct diagnosis of intracranial complications before operation. This case illustrates that point very clearly, as the patient gave no symptoms of which has been pointed out by Sondern to be a good indica- brain abscess prior to its discovery at the time of operation. The dizziness complained of, I think, can be explained by the extreme intratympanic pressure, the mental dullness was attributed to the influence of the morphin which she had been taking.

We also have in this case an illustration of the importance of weighing very carefully all postoperative symptoms and eliminating all possible sources of their development before deciding that they are dependent upon an extension of the original infection, and still further complicating the case by an unnecessary operation. I feel sure that had I assumed from the misleading postoperative symptoms present that there was further brain involvement, and made an exploratory operation, I would have jeopardized my patient's chances of recovery.

I want to again call attention to the value of daily blood counts as an aid in arriving at a correct diagnosis in cases where there is much apprehension and puzzling symptoms. Especially is it of importance in postoperative cases of septic intracranial involvement, which may, as in this case, develop alarming symptoms, dependent upon some intercurrent condition. It was of the greatest value to me in this particular case, as I would have been strongly inclined to reopen the brain wound, in view of the alarming symptoms, had not the blood counts taken daily shown a diminishing polynucleosis and a steady leucocytosis, thus shortening the resistance line, tion.

My method of packing the abscess cavity might perhaps be criticised, in view of the experience of many of our best operators, but my decision to use the iodoform gauze was based upon the fact that it was a small cavity, showing a tendency to contract, and that I was able to enter it directly along the route of the infection; therefore, in all probability the brain coverings were thoroughly amalgamated over this area, thus minimizing the possibility of a septic meningitis. The result in this case was a perfect recovery.

XXXIII.

SINUS THROMBOSIS FOLLOWING NECROSIS OF PERIOSTEAL FLAP.*

By Otto Glogau, M. D.,

New York City.

Mr. P. D., 40 years of age, came into my clinic at St. Mark's Hospital Dispensary, on November 5, 1912. He was complaining of severe pains and entire lack of hearing in the left ear. The pains lasted for two weeks. The ear was running, a small perforation at the posterior superior quadrant of the drum being present. The drum was inflamed and bulging. There was much sensitiveness to pressure over the antrum and at the tip of the mastoid process.

A paracentesis was performed, and the patient was directed to make hot applications and irrigations for two days. After this the ear discharged freely, the pains and the general condition of the patient became worse.

On November 8th he was admitted to St. Mark's Hospital with a temperature of 101.° The next day I performed on him a simple mastoid operation. The usual curvilinear incision was made, the skin and adjacent tissues were divided down to the fascia. I then formed a Leland periosteal flap. This was the first time I had tried Dr. Leland's method, which so very much impressed me at the demonstration in Boston, before the Ninth International Otological Congress. In forming the periosteal flap the rules laid down by Dr. Leland in his paper were closely adhered to: "The classic curvilinear incision is made from the level of the upper insertion of the auricle to the mastoid tip, one-fourth inch behind its posterior border, dividing the skin and subjacent tissues down to the fascia. The tissues are then dissected forward to the cartilaginous meatus, and backward as far as it is possible to go, by lifting up the skin, say one and one-half to two inches. The flap is then to be formed of the periosteum and fascia and the connective tissue between them. To this end the knife is introduced at the posterior border of the tip of the process at right angles to the surface down to the bone, the tip of the left index finger being used as a guide to prevent injury to the emissary vein. From the tip the knife is carried upward and backward along the posterior border, then backward horizontally for one-half to one-third inch or more, then directly upward to the temporal line, then forward to the posterior border of the external meatus, but about one-half inch above it. This periosteal flap, including the attachment of the sternocleidomastoid muscle, is then removed from the bone with a strong elevator, which sometimes is not accomplished without considerable difficulty. We then have a flap of considerable thickness with a broad vascular attachment anteriorly." (George A. Leland, Boston: "A Periosteal Flap in Mastoid Operations," Transactions Ninth International Otological Congress, Boston, 1912, p. 259.)

---

*Read before the New York Academy of Medicine, Section on Otology, February 14, 1913.

The periosteal flap having thus been formed, the mastoid cavity was opened. At the first stroke of the chisel the sinus became apparent, surrounded by pus and granulation tissue. The protective granulation wall of the perisinus abscess had apparently left the sinus intact; its color was normal, there were no symptoms pointing to a thrombus. All cells of the mastoid process had been destroyed and replaced by pus and granulations. The mastoid cavity was thoroughly evacuated, and also the granulations around the antrum removed and the antrum itself enlarged. The cavity was then washed by warm normal salt solution.

Leland's periosteal flap had been formed before it was ascertained whether there was an exposure of the sinus present or not. Although Leland had applied his method only in uncomplicated simple and radical mastoid operations, the writer decided to make use of the periosteal flap present, in order to test its applicability in complicated mastoid conditions. Following the instructions of Dr. Leland, the flap "was brought down to cover the aditus, and a bit of the tip to lie against the inner wall of the antrum. The tip was anchored in the proper position in the aditus by the rounded end of a thick gauze wick, packing it tightly down; and the rest of the flap against the posterior surface of the thin meatal wall. The outer part of the cavity was very lightly packed with gauze, and the lips of the skin wound sutured to bottom, leaving a length of about an inch through which the packing protruded." Special care was taken to keep the flap away from the exposed sinus. Four days after the operation, at the first dressing, the loose gauze, but not the anchor, was removed; the temperature began to rise and reached on the fifth day 104.4.° The patient was suffering from very severe pains on the left side of the head, and especially upon pressure alongside the inner margin of the sternocleidomastoid muscle. He began to cough and to feel unpleasant sensations in the chest. The next day the temperature dropped down to 101,° and the patient suffered from exhaustive perspiration. On November 17th (eight days after the operation), the temperature rose to 105.° This rise in temperature was preceded by a severe chill lasting for eight minutes.

As all symptoms pointed to the presence of a sinus thrombosis, the writer decided to explore the mastoid cavity. On November 17th an incision was made through the old wound and the cavity exposed. The periosteal flap was found dislodged from the antrum and covering the sinus; it had apparently become entirely necrotic. The sinus showed grayish discoloration and felt hard on palpation; pulsation could not be detected, nor did dilatation take place above the compressing finger. By removing the intervening bone the sinus was then exposed downward to the jugular bulb and backward to the torcular, and was found to be discolored and hardened to that extent. An exploring needle was inserted and no blood could be aspirated from the sinus. The sinus wall was then incised through its entire thickness. No bleeding occurred, as the sinus was found to be filled with a thrombus reaching both to the torcular and to the bulb. The writer decided to ligate the jugular vein prior to the removal of the thrombus. The mastoid wound was therefore temporarily covered with gauze and the neck antiseptically cleaned. The incision was made alongside the anterior margin of the sternocleidomastoid muscle, extending from the level of the angle of the upper jaw to the cricoid cartilage. After dividing the superficial tissues by the scalpel, the deeper structures were reached by blunt dissection and the common sheath opened. The jugular vein was then exposed, after being carefully separated from the pneumogastric nerve and the carotid artery. The vein was apparently filled with a blood clot down to the entrance of the facial vein; half an inch below the latter the jugular vein was ligated in two places, one inch apart, and severed. The jugular vein was then exposed high up towards the jugular bulb and ligated as near as possible to it; after ligating the tributary veins the thrombosed portion of the jugular was removed. A rubber tissue drain was then inserted from the upper to the lower end of the wound and the incision closed above it. The packing was then removed from the mastoid wound and the thrombosed sinus gently curetted down towards the bulb and backward towards its torcular end, until free flow of blood occurred. The lips of the opened sinus were compressed by gauze and the mastoid wound packed.

The next day the corresponding side of the face became swollen and edematous; this condition disappeared after five days. The temperature dropped immediately after the operation and with slight remittances soon approached the normal. The wounds on the neck and ear healed rapidly. The patient made an uneventful recovery and was dismissed from the hospital eleven days after the removal of the thrombus. The mastoid wound closed three weeks afterwards by granulation.

About one month later the patient developed an acute mastoiditis on the right ear and was operated upon by the writer. The wound was closed by subcuticular suture with small secondary counter drainage.

The patient was not presented with the idea of reflecting generally upon Dr. Leland's method of periosteal flap formation. Although strictly formed after Dr. Leland's instructions the flap did not adhere to the antrum, but became dislodged and encroached upon the sinus. Dr. Leland himself had a few cases where "the flap had pulled away from the aditus." He ascribes this accident to the shortness of the flap, and advises "to tear it away from its new attachment and repack it over the antrum." It is difficult to decide whether the periosteal flap in this case became necrotic from lack of proper nourishment or from infection by the granulations covering the sinus. However it may be, the sinus thrombosis was doubtless due to necrosis of the periosteal flap. The writer has no experience with the periosteal flap in uncomplicated mastoid operations. The above case teaches merely that the periosteal flap should never be made use of where there exists an exposure of either the sinus or the dura.

XXXIV.

A CASE OF PARALYSIS OF THE ABDUCENS NERVE ASSOCIATED WITH ACUTE MASTOIDITIS.*

By Harold Hays, M. D.,

New York City.

During the past winter, Dr. Charles E. Perkins reported six cases of paralysis of the abducens nerve associated with a suppurative condition of the middle ear. He also collected ninety-five cases from the literature. In all of his cases the paralysis disappeared within a few months. Perkins claims that the paralysis is often due to the narrowing of Dorello's canal "from inflammatory thickening of its walls, or the thickening may arise from edema caused by inflammation in the neighboring dura or bone." This narrowing, however produced, would result in pressure upon the nerve or abducens paralysis; or the inflammation may extend to the nerve itself, causing neuritis with consequent loss of function.

In December, 1911, Dr. John R. Page reported a case of sinus thrombosis followed by abducens paralysis.

I wish to add the following case to those already recorded: J. D., 17 years old, was admitted to Dr. Robert Lewis' service at the New York Eye and Ear Infirmary, September 7, 1912. Immediately after sea bathing, a month before admission, the patient began to have pain and a ringing sensation in the right ear, and the following day the ear began to discharge. This discharge was continuous up to the time of admission with very little pain. He had considerable headache over the right temporal region. There had been no previous trouble with the ear.

Examination of the right ear showed a perforation at the middle of the posterior quadrant, and the canal was filled with pus. There was no mastoid tenderness. The left ear was negative. General physical examination, negative. Temperature, 100.8°; pulse, 92; respirations, 20. September 9, 1912, the patient has had constant pain on the right side of the head. Diplopia developed six days previous. There occurred a paralysis of the right rectus muscle. Nausea and vomiting came on two or three times a day for six days. There was no mastoid tenderness. Free purulent discharge from canal. Knee-jerk reflex diminished on right side. Kernig sign negative. Rhomberg and Babinski reflex negative. Slight papillitis in right eye. No nystagmus. Temperature, 105°; pulse, 112; respirations, 28. Urine examination showed slight trace of albumin. Examination of pus from canal showed principally epithelial cells. Blood count: White blood corpuscles, 10,000; red blood corpuscles, 5,400,000. Differential count: Large mononuclears, 4 per cent; small mononuclears, 17 per cent; polynuclears, 79 per cent. Blood culture (15 cc. of blood) showed two colonies of staphylococcus aureus in twenty-four hours. X-ray picture of mastoids showed that they were diploetic and both sinuses well forward. On the right side was a small oval shadow about half way down below the knee and posterior border of the sinus, which was either a cell or perisinus abscess. (This proved to be a small cell over a large opening of the mastoid vein into the sinus.) Wassermann negative.

---

*Read before the New York Academy of Medicine, Section on Otology, February 14, 1913.

On the afternoon of September 9th a simple mastoidectomy was performed, on account of the paralysis of the sixth nerve, the high temperature, the peculiar symptoms and because of the X-ray finding. The mastoid was found to be quite sclerotic with only a few fairly large cells which were filled with granulations. No free pus was found. The sinus was intentionally exposed throughout a great part. No dura was exposed. The antrum was opened and curetted out, the tip was completely removed. The wound was partially closed with clamps and packed with iodoform gauze.

A lumbar puncture was performed on the table; 15 cc. of clear fluid under slight pressure was withdrawn. This contained no traces of albumin. There was no appreciable sediment in the contrifuged specimen. The culture of this fluid was negative after twenty-four hours.

The following day the temperature was 101.2°; pulse, 84; respirations, 24. September 12th, wound cleaned. Slight discharge from canal. Patient says diplopia is less marked.

There is a well marked nystagmus on rotation of the eyes to the left. There is no nystagmus to the opposite side. With an object held at a distance of about a foot from the eyes, diplopia is noticed as soon as the object moving from left to right passes the midline of the body. Urotropin was given every four hours. Examination of the eyes showed that the papillitis had practically subsided. September 26th, patient complained of slight headache. September 30th, distinct exophthalmos on right side. There is some tenderness over the right frontal sinus. X-ray negative. October 24th, wound healed and patient has left the hospital.

Perkins in his paper remarks that intense headache, "especially in frontal region, severe pain in the eye or deep in the orbit, neuralgia of the fifth nerve, or paralysis of some of the muscles supplied by it, are symptoms, one or more of which have been noticed in fifty-five out of ninety-five cases reported." This means that there is some interference with the Gasserian ganglion by the process going on at the apex of the petrous bone. Gradenigo first called attention to the severe pains in the frontal and parietal region associated with purulent discharge from the ear and abducens paralysis, and therefore this is called Gradenigo's syndrome.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

Meeting of January 10, 1913.

Report of Cases of Inflammatory Diseases of the Labyrinth.

DR. WENDELL PHILLIPS said that the cases presented represented the work done by himself and his assistants in hospital and private practice during the past two years, and were presented just as the histories read. In the light of more recent improvements in the recording of histories, some of them showed errors; the more recent records, however, were more carefully made, and consequently were more readable and understandable.

Simply the reports of the cases were presented, without comment, in order that the members might have a faithful statement of what had been done.

Lantern Slide Demonstration of Section of the Temporal Bone, With Special Reference to the Anatomy of the Labyrinth; Also Exhibit of Microscopic Specimens Demonstrating the Histology of the Internal Ear.

BY ISIDOR FRIESNER, M. D., AND ALFRED BRAUN, M. D.

(*By Invitation.*)

Paper: Model Reconstruction of the Vestibular Apparatus.

BY EUGENE LEWIS, M. D.,

DUBUQUE.

(*By Invitation.*)

DISCUSSION.

DR. KERRISON, in opening the discussion, said that in the number and character of the cases reported, this meeting was one of the most interesting and notable which the section had held.

Considering the serious character of the cases reported by Dr. Phillips, the results, taken as a whole, were exceptionally good and a subject for congratulation. It was worthy of note that in the fatal case reported by Dr. Phillips—i. e., a case of diffuse suppurative labyrinthitis in the latent stage—a radical operation had been performed without operating upon the labyrinth. This patient died of meningitis. Dr. Kerrison said that he had himself published the report of a somewhat similar case in which he had followed the same course, his patient dying later of cerebellar abscess. Personally, he was firmly convinced that in the long run it would be found safer not to do a radical operation upon patients suffering from any stage of suppurative labyrinthitis without at the same time providing surgical drainage of the labyrinth.

In two cases which recovered without operation upon the labyrinth, Dr. Phillips reported having found a fistula leading into the vestibule, into which a tympanic probe could be introduced and brought out through the open oval window. Drainage of the vestibule had therefore been provided by the necrotic process. Recovery in these two cases without operation would seem to support the proposition that adequate drainage is the chief desideratum in suppurative labyrinthitis. Dr. Fowler has reported an instructive case of long-standing middle ear suppuration, in which the tympanum was to a very considerable extent epidermized; yet later a large sequestrum, including parts of the labyrinth, came away. Such a case provides a graphic illustration of the possible progress of a necrotic process beneath an epidermal covering; and it supports the argument that a radical operation is not a safe procedure so long as a suspected focus of infection within the labyrinth remains undrained.

The speaker said that the cases reported by Dr. Kopetzky were very interesting, and the good average results seemed due to the good judgment the doctor had shown in determining which should and which should not be operated upon.

Dr. Kerrison said that he had listened with a great deal of interest to Dr. Lewis' lecture. To review the physiology of the vestibular apparatus in terms sufficiently clear to be easily followed was in itself something of a feat. Dr. Lewis had not only accomplished this, but had held the interest of his hearers throughout. The points in which Dr. Lewis differed somewhat from the accepted views of vestibular physiology—e. g., in his eplanation of the production of certain forms of rotation after-nystagmus—were exceedingly interesting, and he hoped to see the lecture preserved in permanent printed form.

Dr. George E. Davis congratulated Dr. Lewis on his ingenious model of the vestibular apparatus and his able discussion and demonstration of the vestibular functions. He agreed with what Dr. Kerrison had said about discussing the paper, as the subject was a very difficult one to conceive or follow. He could not understand why Dr. Lewis' apparatus did not demonstrate the mechanism of the functions of the vertical canals as taught in the Viennese clinics in a like manner as he claimed it demonstrated the mechanism of the functions of the horizontal canals as taught in the same clinics. It is a well recognized law that the momentum of the endolymph currents produce a more effective and a less effective reflex movement, depending on the direction of the currents, whether from the canal to the utricle or from the utricle to the canal. In the horizontal canals the more effective or preponderant movement or reflex occurs when the current is from the canal to the utricle; in the vertical canals the reverse is true. Now it matters not whether the two labyrinths are one organ, as Dr. Lewis contends, or whether they are separate organs and act in concert, in which latter view I agree with Dr. Kerrison, the above law still obtains and applies in the same measure to the vertical canals as to the horizontal canals. Furthermore, whether the anterior vertical canal of one side acts in concert with the posterior vertical canal of the opposite, or whether the two anterior vertical canals act consensually with the two posterior vertical canals, as I understand from Dr. Lewis' model and demonstration, the above spoken law, I believe, still holds and the Viennese teaching is rather confirmed than refuted.

Moreover, a careful study of the anatomic considerations underlying the physics of vestibulation and the function of the semicircular canals, inclines me to the belief that the maculæ of the utricle and saccule, and the ampullæ of the semicircular canals, are primarily receptive organs and correspond to the end sense organs of the cochlea in this respect. Therefore, if the function of the vestibular apparatus is dependent on physical laws, the principles of its mechanism may be analyzed.

The peculiar arrangement of the labyrinthine sense organs in the cristæ ampullæ of the semicircular canals and in the maculæ of the utricle and saccule bears a basic relation to the mechanism of their physical reaction to stimuli. When we consider that the portion of the vestibular nerve which arises in Deiter's nucleus communicates fibers to the nuclei of the motor nerves of the eye muscles and to the motor nuclei of the spinal cord, we can understand that tonogenesis is due to stimulation of the central nuclei and that the function of the labyrinth is for the most part to receive and transmit said impulses, sensations or stimuli.

I may say that, also on account of anatomic consideration, it is a plausible theory that the maculæ of the utricle and saccule interpret motion and direction in straight lines, while the cristæ ampullæ interpret rotatory motion in the several directions of the planes of the semicircular canals in the three dimensions.

The chief sources of stimulation of the vestibular apparatus are motion and gravitation. Motion results from variation or position, and this change of position produces the variation in pressure, stress or impact of the endolymph currents on the cristæ ampullæ and the maculæ. This impact or stress causes pressure stimuli, which are received by the hair cells of the cristæ and the maculæ, which in turn convey them along the nerve fibers to the brain and spinal cord. Gravitation explains the source of constant pressure stimulation by the otoliths on the maculæ, which receive and conduct them thence to the brain, which in turn perceives them and exercises the necessary tonogenesis to maintain equilibration.

An analysis of the labyrinthine functions, based on physical laws, favors the argument that the major function of the vestibular apparatus is to receive and regulate tonus impulses which are conveyed thence to the central tonus nuclei where the tonus impulses are developed. Von Cyon goes so far as to say that the destruction of the labyrinths would not destroy muscle tonus, but would increase it by removing the natural inhibitory action, which he claims is one of the most important functions of the vestibular apparatus.

Shambaugh expresses an opposite opinion to von Cyon and says he cannot accept the view that Deiter's nucleus possesses automatic tonus centers, but that the voluntary muscles are controlled by the tonus impulses from the labyrinth.

Between the extremes of the von Cyon and Shambaugh theories as to the origin and development of muscle tonus impulses and their influence on equilibration—or the adjustment of the individual to space orientation—or the recognition of that adjustment, I believe there is a middle ground.

Dr. Bernard Sachs said that he did not quite understand why he had been asked to speak on this topic, but was inclined to think that the chairman meant to show how a man could be made dizzy without rotation, by being compelled to listen to so much learning. As a neurologist, however, he would like to refer to one or two points in the paper. Dr. Lewis' presentation of the subject was extremely interesting; it was not difficult for the speaker to conceive of these two portions of the vestibular apparatus acting as one. It was of great value to have this subject presented so clearly as Dr. Lewis had done it. The vestibular apparatus is acquiring great importance just now, not only for the otologist but for others also, and this should be remembered by the otologist, for it is going to enlarge his own field of examination and research. The vestibular apparatus has not been insisted upon so far as a part of the statotonic apparatus. To this belong the entire skin of the body, the eye and the auditory organ; relations in space are determined by these three various senses, and the vestibular apparatus is an important contributing factor in this arrangement. The most important organ of the apparatus is not the labyrinth but the cerebellum, for all these paths are finally centered in the cerebellum, and the vestibular apparatus is one only of the three chief component parts of the larger apparatus ending in the cells of the cerebellum. There was one point which Dr. Lewis spoke of which the speaker would like particularly to commend—that was that the labyrinth should be regarded as one of the three important factors of the statotonic mechanism. It was perfectly true that disease of the labyrinth influences the status of the peripheral muscles; that was a point which the otologists should bear in mind, and so make a routine examination of the deep reflexes of the body and of the various muscles, as this would give much assistance.

The vestibular apparatus is only a part of the larger spinal and cerebellar apparatus, and very much the same symptoms result if any of the three parts of the huge mechanism is disturbed. The difficulties of diagnosis are increased by this. We have always supposed that hypotonus would go with purely cerebellar disease, and in cases where we found such affection or modification of the tonus we have always been ready to diagnosticate a central and not a peripheral lesion. Labyrinthine disease may be accompanied by hypotonus; and if so, the caloric tests must be depended upon to give a clue as to the actual site of the trouble.

Dr. Danziger said that he wished to take exception to the statement of the reader of the paper that we cannot test the function of one labyrinth. If we rotate the patient, we certainly set the endolymph of both labyrinths in motion, and the fluid will strike the hair cells of the crista, the ones of higher valency in the labyrinth toward which side we rotate, and the ones of lower valency in the other labyrinth. The sum total will give the intensity of the nystagmus. Supposing we turn first to one side and then to the other—the difference of the intensity of the nystagmus will show the functional change of one or the other labyrinth. For clinical purposes, we can, therefore, test either labyrinth for its functional state.

Dr. Strouse said that he wished to call attention to one point in regard to the falling of the patient. Dr. Lewis had said that the falling was not caused by the vestibular disturbance, but by the disturbance of the eye caused by the nystagmus, and the patient falls toward the slower complement. Dr. Strouse said that he could not reconcile that with the conditions as they exist, otherwise in horizontal nystagmus the patient would fall to one side or the other. If there is a vertical nystagmus, according to that he ought to fall backward or forward; and if there is a rotatory nystagmus, he ought to turn cartwheels to one side or the other.

Dr. Friesner said that he did not believe that the falling was due to the ocular impression; that, of course, could not be true, for the falling resulted from the reaction movements. These are distinct although not pure reflexes, and result from the stimulation of the vestibular apparatus. Moreover, the falling occurs even when the eyes are closed. He said, further, that he agreed with the others who had previously discussed Dr. Lewis' paper, that this theory could be discussed only after careful study and consideration. If, however, this theory depended upon the absolute parallelism of the corresponding canals, it must fall to the ground, since Schoenemann has shown, from the examination of some 500 or 600 corrosion specimens, that considerable differences exist between the two sides. These differences sometimes amount to twenty degrees.

Dr. Phillips said that the microscopic slides shown by Drs. Friesner and Braun were in the laboratory of the Manhattan Eye, Ear and Throat Hospital, and they would be very glad to show them to the members any Tuesday, Thursday, or Saturday afternoons. As to the remarks which had been made concerning the cases reported, he only wished to repeat that the cases had been presented from the histories without any comment.

He felt that he had just begun his study of these purulent conditions, and he hoped that the eighteen or twenty cases presented this evening would prompt some of the other members to report similar cases until two or three hundred were recorded. In only one of the cases which he had reported would he act differently, had he to do it over again. This was case 3 in his series, wherein he had performed operation early. In that he believed he should have done the radical and labyrinthine operation or should have let it alone.

All of these cases, with one exception, were complicated with acute purulent otitis media; every one was a chronic case. There may be a difference in treating chronic and acute cases.

Dr. Fowler said that the case mentioned by Dr. Kerrison was not healed, but was discharging posteriorly. Epidermatization had taken place only in the middle ear, so there was very good drainage. In regard to the diagnosis between suppurative and serous labyrinthitis, he would suggest to the members the use of a band around the neck, in order to congest the head. He hoped some day to tell the section of some experiments he had made with this expedient, as they were very interesting. It stands to reason that increase of pressure in the skull is bound to influence the pressure in the normal labyrinth; it also markedly affects tinnitus in many persons.

In some of these cases the tinnitus can be brought on or stopped at will by using this constriction band.

In acute suppurative disease of the static labyrinthitis (during the onset) the constricting band will markedly increase spontaneous nystagmus, whereas in the nonpurulent cases it appears to have little, if any, effect, or distinctly diminishes the nystagmus.

Tinnitus due to inflammation of the cochlea is increased or diminished, or unaffected, by a constricting neckband, depending on the ability of the band to increase, diminish, or not affect the iritating effect of the tissues responsible.

NEW YORK ACADEMY OF MEDICINE,

SECTION ON OTOLOGY.

Meeting of February 14, 1913.

Sinus Thrombosis Following Necrosis of the Periosteal Flap*

By Otto Glogau, M. D.

Traumatic Atresia of Auditory Canal With Mastoidal Complication.

By Ernest Danziger, M. D.,

Report of a Case of Paralysis of Abducens Nerve With Mastoid Complications.†

By Harold Hays, M. D.

Report of Removal of Fibroma Which Excluded the Auditory Canal.‡

By M. D. Lederman, M. D.

DISCUSSION.

Dr. Haskin, referring to Dr. Glogau's case, said that the doctor had spoken of Dr. Leland's flap. The speaker had seen that method used very successfully. Dr. Glogau had apparently neglected to consider the fact that he had exposed dura and sinus. No one doing mastoid surgery should ever attempt to put a flap of any kind on exposed dura. Dr. Glogau had also spoken of having opened the sinus and curetted the jugular bulb. That is a thing that is no longer spoken of, as it is very difficult to reach the bulb without a most extensive exenteration.

Dr. Leland especially states that in attempting to make use of his periosteal flap a complete exenteration of all diseased bone must be performed. Dr. Glogau admits that in opening his sinus he had to remove more of the bone. Three things might have caused the sloughing of the flap: First, not enough blood supply at the pedicle or too much handling of the flap. Second, too light packing. Third, septic infection. If not misunderstood, the doctor says that the gangrenous flap had slipped down under the packing. With the pedicle attachment above, it is hard to see how such a thing could occur.

---

* See page 509. † See page 514. ‡ See page 431.

Referring to Dr. Lederman's case, Dr. Haskin stated that Dr. Sluder says he never does a tonsillotomy without having previously made a blood examination to determine the time of blood clotting. When it does not take place within a minute and a half, Dr. Sluder refuses to operate.

Dr. Cocks, referring to Dr. Lederman's case of hemophilia, said that it was always easy to criticise such cases after one is through with them, but that a case with a marked history of hemophilia should have been tested for coagulability before operating. In ear and throat surgery the practice of the general surgeons should be followed in this respect. In cases of jaundice they give blood serum subcutaneously or intravenously, and bring the blood up to normal before operating. If that had been done in this instance, Dr. Lederman would have been saved much trouble and anxiety.

Dr. Gruening said, in regard to the case of Dr. Hays, that the diagnosis of papillitis or optic neuritis should not be made in connection with some of the complications of purulent otitis. A true inflammation of the optic nerve or its head, an optic neuritis or a papillitis, only occurs when the aural disease is followed by meningitis. The diagnosis of optic neuritis would signify that the patient is beyond hope. What is really observed in many forms of otitis media and its complications is an edema of the papilla, the socalled choked disc. The fact that the edema disappears after the removal of the purulent focus, shows that it is not an inflammation.

Dr. Seymour Oppenheimer, referring to Dr. Haskin's quotation from Dr. Sluder, said that he had read the paper, and had wondered at the statement of Dr. Sluder's, that when he does not get coagulation within one and a half minutes he refuses to operate. It would seem to be a typographical error, for if he refused to operate under such conditions he would not operate often. It has been his own experience that it is quite within the limits of normality for coagulation not to take place within five or six minutes. In regard to calcium lactate, in a series of cases in which he had used it and tested the coagulability both before and after its use, he did not find that the calcium lactate influenced the condition in a single instance. It is probably of value only in distinct hemophiliacs, and not with normal individuals.

Dr. Kenefick asked if he was correct in understanding that Dr. Lederman's patient was a female. That point was of interest, for it is stated in the books that females do not bleed, that they simply transmit the hemophiliac diathesis, and the male members of the family do the bleeding. Relying on the strength of that fact and concealing the family history, a physician recently had referred to him a young girl of about fifteen years of age, from whom he removed adenoids. Following the operation she had all the characteristics of a hemophiliac, and the bleeding could not be controlled until after the injection of 100 cc. of horse serum. It seems, therefore, that the statement that females do not possess the hemophiliac characteristics cannot be depended on.

Dr. Felix Cohn said that in the discussion one of the gentlemen had referred to the coagulation test. It has been his practice for several years, since he has been making enucleations of the tonsils, to have the coagulation time taken in every case before operation. The enucleations were always performed under narcosis and only on hospital patients. The time of coagulation varied from four and a half to eight minutes, the average being about five to six minutes. The tests were made with a drop of blood on a slide, and the time taken for coagulation was carefully noted. The results have been uniformly excellent. It is a question, however, whether in the case referred to by Dr. Lederman, even a coagulation time test could have absolutely safeguarded the patient, inasmuch as there are cases of hemophilia where the coagulation time is not over prolonged, so that even the making of the coagulation test will not always guard the operator from the possibility of operating upon a hemophiliac.

Dr. Glogau said that in operating on the case reported he had followed Dr. Leland's technic closely. The sinus at the first operation was evidently perfectly normal; the flap was put in the direction of the antrum, and care was taken to keep it away from the sinus. It was kept in place by gauze. It became necrotic by not being nourished properly. It was not fair to claim that it was due to poor surgery, for the operation was performed lege artis and the sinus was normal before making the flap; besides special care was taken not to bring the flap into connection with the sinus. In regard to the criticism of what he had said about the curetting of the bulb, he might have been misunderstood. He simply said the sinus was curetted down towards the bulb. In the making of the flap he had followed Dr. Leland's directions very carefully.

Reply to Dr. Danziger, Dr. Glogau said: The jugular vein was thrombosed only to the entrance of the facial vein, and he was ligating below the latter. If the jugular vein would have had time to thrombose further down, the edema of the face might have occurred even without ligation of the jugular vein.

Exhibit of Dissected Temporal Bones.

Dr. William Henry Haskin: Diseases of the labyrinth have become well recognized of late and much is written about them. It seems to the writer as though a more thorough knowledge of the actual anatomy of the parts is one of the most important factors in undertaking any operative measures. To read many of the numerous articles, both in magazines and in books, it would appear to be a very simple thing to open the labyrinth and remove it according to Jansen's, Neumann's, Richards', and other methods, but the writer in presenting these fifty specimens desires to emphasize the difficulties attached to these operations and to warn against rashly undertaking them without a complete knowledge of the many relationships that one may encounter. This work should never be attempted save by those who actually know their anatomy and are prepared for emergencies.

It is well known that no two mastoids are ever exactly alike, but it is impossible to describe in writing the great differences in the bones; this knowledge can only be learned by carefully studying a series of specimens. It has been an invaluable experience to the writer to open each of the specimens presented. In these specimens the mastoids have been exenterated, and they show a great variety of forms, but one cannot appreciate the difficulties of cleaning them out unless he actually does the work himself. Some were composed of enormous cells throughout, extending into the zygoma (Nos.

96, 77, and 76), while others were dense throughout (Nos. 94, 73, and 72); and between these were all the varieties described by Mr. Arthur Cheatle before the International Otological Congress, in Boston, on August 12, 1912, and more besides.

In examining the specimens it would be well to do so systematically, for instance:

1. Shape of mastoid, depth, height and width.

2. Position, relation and direction of both the lateral sinus and the jugular fossa, which are colored with black ink and show many variations. Entire sinus and fossa lie in temporal bone in specimen D.

3. Facial canal. Size, relation to external semicircular; to posterior semicircular; to posterior wall of external auditory canal; to jugular fossa; to lateral sinus; note position of geniculate ganglion (Nos. 63, 110), from which chorda tympani passes back into the middle ear. Nerve is represented by strand of silkworm gut, the loop on petrous portion representing where the greater petrosal branch is given off. It is well to note how the nerve descends, in most cases, as far back as the middle of the external semicircular canal. Also note that it would be next to impossible to enter the tympanum from behind and under the nerve in many cases without opening the jugular fossa and destroying the nerves. (Nos. 66, 65, 63.)

4. Note variations in amount of space between sinus and fossa and the floor of the labyrinth. In several of the specimens large cells extend under the labyrinth to the internal auditory canal (Nos. 95, 107, 108), while in others the fossa reaches above the level of the lower end of the posterior semicircular canal. (Nos. 105, 106, 91, 92, 80, 81, 66, 65 and 63.)

5. Note how well these specimens bear out the conclusions of Schoeneman as to the inconstancy of the direction of the planes of the canals and the angles between them; for instance, it is very rare to find the external canal in a true horizontal plane.

6. The canals vary markedly in the size of the canals and of the arcs which they encircle. The superior and posterior vary greatly in their relation to the surface of the petrous portion of the bone, rarely being surrounded by cancellous

bone (Nos. 92 and 91), but generally forming part of the inner cranial wall.

7. The dense bony walls of the canals and the angle cannot be described, nor can they be appreciated by any one except by actual dissection. After cutting out all cellular tissue these semicircular canals were opened with dental burs, wherever possible to reach them, without taking down the auditory canal. This wall has been preserved in order to give the true idea of relations. In some the dense bone was almost one-third of an inch in thickness. In a few the upper bony angle has been cut away to show the interior of the labyrinth (Nos. 74 and 68), and several have had the ridge between the foramen ovale and the rotundum in the tympanum cut out with a small trephine, as advised by Hinsberg in his operation for opening the labyrinth. (Nos. 70, 118 and 119.)

8. Facial nerve. One specimen shows a large dehiscence between the tympanum and the jugular fossa which reaches well above the middle of the tympanum. (No. 64.)

9. Another specimen shows the facial canal in the anterior wall of the lateral sinus (No. 108), the sinus itself in contact with the labyrinth.

10. The position of the silkworm loop appears to the writer to be the probable point of infection in cases of temporosphenoidal abscess, especially when accompanied by facial paralysis occurring in the course of purulent otitis media. It is the point at which a bristle passed upward through the facial canal from stylomastoid foramen will always appear showing the direct path. To reach the internal auditory meatus it must be bent backward at right angles.

11. Shows sinus in contact with posterior wall of canal for one-half inch, with large cells above and below. The upper cortex over the antrum was very dense to a depth of half an inch. (No. 62.)

12. Nerve, bulb, sinus and geniculate fossa are very close in Nos. 63 and 117.

13. Lateral sinus forms part of basal cranial wall in its horizontal position.

It is unsatisfactory to have so little time for inspection of specimens at our section meetings, for such study is of very great value, but requires time. I would suggest that an entire evening be devoted to this subject, and that members having collections be asked to have them on hand. It is very difficult for most of us to obtain bones, so that an opportunity to see a large collection would be of great value to all, but especially to those who have none. With the enlargement of our present building, it could probably be arranged to have a museum of anatomy if the members would present collections or even loan them to the academy. Such a collection would be of inestimable benefit to all generations and could be made a great credit to our profession, for we have very few anatomic exhibits in this city, that are ever heard of, at least, and it is very difficult for any one to study the many variations in anatomy.

These variations cannot be called abnormalities, as there is no such thing as a normal perfect bone, each one differing in some way from every other one.

Paper: The Enhancing of Auditory Acuity by Psychologic Methods.

By Wm. J. Maloney, M. D.

DISCUSSION.

Dr. Gruening said that he had been asked by the chairman to open the discussion of this paper, although he had not known its character beforehand. Of course the whole subject was largely academic. There was no record of any improvement in deafness by this method. Psychology can go hand in hand with medicine and pedagogy, and can give and derive valuable suggestions. Psychically deaf people are not often observed by the otologist. The psychiatrist may see them more frequently.

He was not willing to endorse the opinion that lip reading is harmful. On the contrary, he had found it a great aid in many cases. Very often when glasses are prescribed for people who are deaf, their hearing is much improved. The deaf who are proficient in lip reading often move in society with ease, and derive much happiness from the facility of their intercourse with other people.

To compare a deaf person with one condemned to solitary confinement is not logical. A person in solitary confinement does not see or hear anything; is dead to the world, dead to himself. Psychologically there are variations in men: the optic and acoustic types. Some live with their ears and learn from what they hear; others see pictures of things, everything is visualized. A conversion from one type to the other is possible and usual. The deaf learn to see better, the blind to hear better than before.

The doctor closed by saying that although he disagreed with the writer of the paper in regard to lip reading, yet he had been very much interested in the paper, and had enjoyed it very much.

PROF. JOHN DUTTON WRIGHT said that he had been much interested in Dr. Maloney's paper. He had never yet found anything that was a universal panacea, anything that would work for every one in all cases. Many things are valuable in certain cases, and are useless in others. There was much truth in what Dr. Maloney had said, and there were elements of great value in his theory.

The chairman had doubtless invited him (Mr. Wright) to take part in this discussion on account of his being known as an educator of the deaf by the lip reading method, and perhaps expected that he would combat anything Dr. Maloney might say against lip reading. It would probably be found, however, if one got down to the basal facts, that he and Dr. Maloney were in perfect accord. He had always insisted that every faculty should be exercised to its greatest extent. He had taught that doctrine throughout his twenty-five years of association with deaf persons—that the possession of hearing, no matter how slight it might be, was their most valuable possession, and that every exertion should be made to use it to its greatest extent.

In his school there are many children who came supposedly totally deaf, and would indeed be deaf for all practical purposes, if allowed to pass their lives under ordinary conditions. In many instances, however, these children have been found to have a residuum of hearing, and the educative problem was how to make the most of that residuum. The greater problem, however, was how to develop the brain to understand the impressions received. You have to teach these children how to hear, and it can be done after three or four years of patient effort. Some one may then come along and remark on the wonderful change, and yet the perception of sound may have been tested before and after the teaching, and found to be absolutely unchanged. The child has simply had its intelligence developed; the ability to interpret the imperfect sounds. He has acquired the intelligent interpretation of sounds into ideas, and apparently hears much better than before.

One will not be successful in teaching the lip reading process unless it is done as a means of getting ideas. Properly taught, the perception of what is said should be just as unconscious as that of the normally hearing person. These people are simply reaching for the thought that is conveyed, and they are ignorant of how to get at it. When a pupil says: "You speak too loud; I heard what you said," and when the teacher knows the range of hearing of that pupil and that he did not hear, the pupil should not be contradicted, for the ideas are coming to him through a new sense without his knowing it, and that is a great encouragement.

Dr. Maloney says that lip reading in those who are not totally deaf is a misfortune and is injurious; Dr. Gruening says it is not. Both are right. It is possible to teach lip reading in such a way as to do the person an injury. It is also possible to teach people lip reading and to preserve every vestige of hearing, and to teach them to make greater use of it. Partially deaf people do not hear everything that is going on around them. They miss a great deal of what is said, and they become indifferent; they become psychically deaf, just as Dr. Maloney has said. They do not pay attention to the sounds, for they do not get enough to catch the ideas, and so they become indifferent and give it up, and like any other organ that is unused, it becomes more and more atrophic, and the psychical deafness increases.

Twenty-five years of teaching lip reading has demonstrated that in teaching pupils one must first experiment with the pupil until the pitch is found where he is conscious of the voice, but cannot possibly understand what is said with the ears alone. Then for a considerable length of time, exactly that pitch of voice must be used which will enable the pupil to be just conscious of the voice, and he will strain every nerve to hear, and the result will be that the sense of hearing is quickened, and the mind rendered more alert, and although the vision does play a considerable part, yet the pupil learns to divide his attention, and the auditory nerve gets a sufficient amount of attention, while the rest is left for the eye.

Lip reading so taught not only enables the pupil to know what is said, but it sharpens and awakens the sense of hearing which was slowly becoming dormant and neglected and running into psychic deafness, so that the person becomes mentally more alert and more interested, and pays more attention to sounds, etc., and gets more satisfaction from living. With a little sound and the help of the eye they can interpret speech without the intense strain which comes from trying to get it without the eye when the ear is imperfectly performing its functions. They say they feel happier, more buoyant, and their physical condition acts upon their mental condition, and the entire apparatus works better.

One thing Dr. Maloney said or inferred which does not seem quite true—that lip reading is the training of the eye. It has been pretty fully determined that the training of the eye is a very minor matter in lip reading. The eye practically performs its functions just as well at the beginning as at the end of this instruction. The pupils are taught to associate ideas with sequences of motion, just as we associate ideas with sequences of sound. It is not the training of the visual sense; it is the training of the power to put ideas with new impressions which have come to them. It is difficult for a normal person to realize the intensity of attention that is required to hear with imperfect apparatus. Most persons have had the experience of trying to hear with an imperfect telephone. If you can imagine your hearing apparatus to be as imperfect as that telephone, you can understand something of the energy that is required to relieve the ear by supplementing it with another organ. If you can relieve that strain you have given the person a fresh outlook upon life.

Mr. Wright further said that in connection with his official duties he had recently had occasion to examine a great many deaf children in schools for the deaf in the State of Massachusetts, and he wished to impress upon persons who have influence the necessity of giving more attention to the development of the hearing in these children, especially the young people in the schools. In every school, and in almost every class, he found children that had some capacity for hearing, the most valuable thing they possessed, which was being ignored—and because it was inadequate they were psychically deaf. They were not physically deaf, in the sense of not being able to hear at all, but they were sufficiently deaf not to have any value or satisfaction from their imperfect hearing, and they ignored the sounds which came to them. Yet, if these children had the proper training, they could be developed into actively hearing people. More attention should be paid to the training of hearing in the schools for the deaf, for there are hundreds in those schools who are being trained as practically deafmutes.

Dr. Maloney was perfectly right in saying that it is desirable to develop the power of hearing to its utmost capacity. Dr. Gruening was perfectly right in saying that if one has not any hearing it cannot be developed; but there are certain fundamental principles which must be kept in mind. When you examine one of these deaf persons and make a chart, and block the islands of hearing which remain, you have to recognize the fact that unless these islands lie in the comparatively narrow range of the speaking voice they are practically worthless. Before the American Association to Promote the Teaching of Speech to the Deaf, Dr. Blake, of Boston, laid much stress on these islands of hearing, but said they were of no value for purposes of communication unless they were in the range of speech. If one wishes to get all around benefit, he must work upon those islands that do lie in the range of the speaking voice. It is the business of the teacher of lip reading to get people into communication with the world. If the island of hearing lies beyond the range of the voice, it will have to be supplemented in some way, and this method of lip reading is a vicarious way of doing it.

He then introduced Mrs. S., who said: "I am only a little hard of hearing, but my ear specialist advised me to take up the study of lip reading. I have not been under instruction long, but I have been surprised to find the effect that this mental effort has had upon my power of hearing. Not only do I understand people better already, but I find that I pay much more attention to the sounds around me. My maid has noticed and commented upon it. I was becoming indifferent and inattentive. I am now much more alert and interested. I listen more intently. I pay more attention to what is said and done around me. I find that I am already relieved a great deal from the nervous strain of deafness. I am less anxious and nervous. I feel greater confidence in myself and therefore succeed better."

Dr. E. W. Scripture said that the reader of the paper seemed to have confused several different kinds of deafness, and that he used the term "psychical deafness" to mean several different things. In the cases he especially described, namely, those who were hard of hearing, his suggestion to omit the direct method known as lip reading was contrary to the experience of all persons who had had any intimate acquaintance with the partially deaf, and also in defiance of the well known fact that the best way to develop the remains of hearing is to cultivate the sensations connected with speech.

Dr. James J. Walsh said that he was surprised that no one had yet recalled the fact that Urbantschitsch of Vienna had done something like this years ago, teaching children who had never heard and were said to be deaf, how to hear. In the course of a few weeks he taught a young person to listen and to hear. The main benefit, of course, was the confidence they get in going around the streets. These people after a while were sometimes able to hear even ordinary speech. The method employed was simply that of standing beside the patient and calling out one sound after another, noting the sounds that got to the ear, and gradually lowering the amount necessary until the attention was educated. In this way he taught them to pick up sounds that they could not pick up before. Dr. Walsh said that he was not so much interested in the otologic side of the subject as in its psychology. In the case of a gentleman who reads with his finger tips, the sense of feeling is so awakened and so keen, that he feels the difference in the letters. He has not had a bit of increase in the amount of sensitiveness, but he has learned to translate the little differences of feeling as they occur, so as to be able to read without any difficulty. Just exactly the same can be done to the eye with regard to lip reading. It is not the eye, it is not the acuity of vision, but the ability to translate impressions which it did not do before so as to get information just in that way. When Dr. Maloney had first spoken of this method, he had referred him to Urbantschitsch's work.

Dr. Walsh said that it had always seemed to him that in the whole system of training one can take every sense and train it in any one. A remarkable instance of this is seen in Helen Keller, who has learned to listen with her finger tips. Most of us have learned to neglect the sense of feeling, and yet she has learned to hear with her finger tips. Can we not take any deficient sense, such as hearing, and train it so as to secure more hearing? Certainly Dr. Maloney's method of blinding the pupil so as to exclude the attention of the eye for a while in order to concentrate the attention upon the hearing was a valuable point.

Dr. Walsh said that he was with Fränkel in Paris when he was first teaching tabetics to walk by having them use their eyes. Dr. Maloney, on the other hand, has them shut their eyes and have them concentrate their attention on their muscle sense. He is applying Urbantschitsch's principle of cultivating attention, and we may look for some interesting results.

Dr. Kenefick said that it occurred to him while Dr. Maloney was reading his paper and bringing out the principles involved, that no mention was made of the time it takes to develop the fibers or sound tracts. It certainly seemed that the time required by Urbantschitsch to make a patient appreciate and repeat the syllable "oo," for instance, or "ee," while he shouted in his ear at the top of his voice at intervals from September until May or June, did not seem a very promising method of teaching the profoundly deaf. One young woman in the Vienna clinic was enabled by this method to hear three vowels during the year that he (Dr. Kenefick) was there as a student.

He had been delighted to hear Dr. Maloney's paper, but in comparison with lip reading, from which all have seen such excellent results, it would seem that for practical purposes lip reading has the advantage at present over the method so ably advocated here this evening.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

Meeting of March 14, 1913.

Report of Sixty-two Cases of Blood Clot Healing Subsequent to Mastoidectomy, Showing the Importance of Exposing and Curetting the Attic of the Middle Ear.

Dr. Hugh Blackwell: The sixty-two cases here reported were undertaken with the view of shortening the duration of healing, lessening the pain of dressing, and improving the cosmetic result of mastoidectomy. The work extends over a period of four years. In about the first thirty of the cases, simple mastoidectomy was performed, and in the last thirty-two, in addition to the mastoid procedure, the attic of the middle ear was exposed and curetted free of infected tissue, without, of course, a disturbance of the ossicular chain.

This was performed by taking down the posterior canal wall to the epitympanic ring and removing the external attic wall, exposing the head of the malleus, body of the incus and its short process. At the conclusion of the operation a strip of iodoform gauze, folded once or twice, is laid down to the antrum and brought out at the inferior angle and the wound sutured down to within about the last one-half inch. A wet dressing of normal saline is then applied. In twenty-four or forty-eight hours the sutures are taken out, together with the posterior packing, which may or may not be replaced. Wet dressings are usually continued for the first week. The canal must be packed tightly at each dressing, and for about the first two weeks.

When the wound shows any tendency to suppurate, it is dried out and packed lightly with iodoform gauze.

Analysis of the sixty-two cases—Adults, 38; children, 14; babies, 10. Infection: Streptococcus, 12; staphylococcus, 3; streptococcus capsulatus, 3; pneumococcus, 2; mixed, 2; unknown, 38; negative, 1; known, 22. Healing duration:

Known, 50; unknown, 11. Perisinus epidural abscess, 19. Subperiosteal abscess, 22. Exposed sinus and dura, at conclusion of operation, 32. O. M. P. C. (2 still wet), 9. Complete B. C., 3. Complicated by subperiosteal abscess simulating furuncle, 3. Erysipelas, 3.

Of the sixty-two cases, thirty-eight were adults, fourteen children, and ten babies under one year of age; of the twenty-two known infections, twelve were streptococcus, three were streptococcus capsulatus. In adults healing took place in about twenty-one days; twenty-three of the cases being cured in less than that time, twenty after that time, five at the end of twenty-one days, and in fourteen the exact time of healing is unknown, but about corresponded in point of duration to the other cases.

Nineteen of the cases were epidural perisinus abscesses; twenty-two were complicated by subperiosteal inflammation or abscess; nine had a chronic discharge from the ear with little or no polypoid granulation tissue except in the attic, the hearing being good. Three of the cases were subperiosteal abscesses opening into the posterior wall of the external auditory canal, and were first diagnosed and treated as furuncles; in each instance they were complicated by a perisinus abscess. Three cases developed erysipelas; all mild cases, one of which was treated as erysipelas for only twenty-four hours. In both the other instances the wounds were doing well at the time of development of this complication, which first appeared some distance away on the face. Dura or sinus was exposed in thirty-two of the cases. None of the cases operated upon died, and in only one was there an operation for recurrence, which took place some two years after the primary operation.

In none of the cases reported has dislocation of an ossicle occurred during the operation.

Quantitative hearing tests have shown that the hearing was no more impaired in the thirty-two cases in which the attic was cleaned out than in the previous thirty. The external auditory canal does not collapse if it is packed snugly at each dressing. In one of the cases of chronic discharge, presented at the conclusion of the bone operation, a radical flap was cut from the canal and the posterior wound sutured, the wound being packed from the canal as in a Stacke operation, no blood clot being used. The result was very gratifying, particularly from a functional standpoint.

Conclusions.—We believe that in a certain number of selected cases of O. M. P. C. with good hearing, presenting evidences of true attic suppuration only, this operation, either with or without a blood clot, or with or without a plastic meatal flap, will preserve the hearing and remove from the ear its potential possibilities of menace.

That when the procedure is employed the duration of healing after mastoidectomy will be materially shortened to within an average period of three weeks in adults. That the dressings are less painful and the convalescence more comfortable. Finally, that the subsequent scar is much less depressed.

DISCUSSION.

Dr. W. S. Bryant said that Dr. Blackwell's paper was upon a most important subject, and one in which he was deeply interested, and that he had watched with much satisfaction the favorable course of Dr. Blackwell's cases. In the later eighties Dr. Henry Lee Morse, Dr. Bryant's senior at the Massachusetts Charitable Eye and Ear Infirmary, gradually reduced the amount of packing in his mastoid wound until there was none. He at first thought it necessary to open the wound widely every day with a director. Later, noting that no pus escaped, Dr. Morse gradually discontinued this practice and finally left the wound alone to heal by first intention as soon as might be. Dr. Morse did not publish his technic and consequently it went unnoticed by the profession. In 1892 Dr. Blake of Boston and the late Dr. Sprague of Providence undertook the blood clot dressing. Dr. Sprague was at that time a general surgeon, had treated the long bones with blood clot dressing very successfully. Since 1892 various men have taken up the procedure in various parts of the country, some securing very good results. He himself has been doing it continuously.

One of the points emphasized by Dr. Blackwell was opening the attic and curetting the middle ear and the removal of all the affected tissue, as far as possible, without disturbing the ossicles. Dr. Blackwell's technic was somewhat similar to that described by Mr. Heath of London, but more like the technic in a paper published by himself (Dr. Bryant) entitled "The Modified Radical Mastoid Operation."

The hearing is quite as good as if the middle ear had not been curetted at all. Dr. Bryant said that he had not thought it well to go as far as Dr. Blackwell had done as a routine, as the result is quite as satisfactory and can be obtained with less risk in most cases. It is quite possible to be a little over-zealous in curetting, with the possibility of dislocating the hammer and anvil.

He congratulated Dr. Blackwell on the results of his work, for they certainly were most creditable.

Dr. Meierhof said that in this method another step forward has been made in the mastoid operation. If one recalls the first mastoid operations done by Schwartze, where a simple opening was made and the mastoid reached, and compares it with the work done today, the advance is impressive, though in those days they seemed to cure most of their patients. Later the operation was widened by a more extensive exposure of the cells, and a point was made of removing the tip, as the tip is often the site of the disease. Then came the removal of all the cells, especialy those around the sinus. Then it was found that the cells in the zygomatic root had to be removed, and still the patients did well. Now comes Dr. Blackwell with this procedure of going far forward into the antrum and exposing the attic horizontally, with no danger to the ossicles, which might ordinarily be supposed to injure the hearing.

Dr. Meierhof said that his experience with this method was very limited as compared with Dr. Blackwell's. Dr. Blackwell had assisted him in several operations, though he did not go so far as Dr. Blackwell advised, but contented himself with opening the mastoid and removing the cells and the tip, and going to the zygomatic arch. With this procedure his results had been very satisfactory as a rule, though in some instances he had been disappointed, as some of the patients had headaches which continued for some time after the closure of the wound. In some instances, also, the contents of the sac which was formed by the breaking down of the blood clot caused signs of tension, and he had been compelled to open up the sac. Still the patients did well and the wounds healed nicely. The leaving out of the packing was a great gain, for the patients dread the dressing very much, especially the first time. He had had no secondaries, the hearing has been good, and the scars have been very shallow, instead of the old depressed scars. Dr. Blackwell was certainly to be congratulated on the results he had attained. Dr. Meierhof said that he was doubtful whether he would follow Dr. Blackwell's example in exposing the attic, in the simple mastoid operation, as he had not found it necessary.

Dr. Felix Cohn said that we must all be indebted to Dr. Blackwell for his presentation, exhibiting the results of primary closure of the mastoid wound in acute mastoiditis. It was not his intention to discuss the paper of Dr. Blackwell. as much as to make a few brief remarks on the subject of primary closure of the mastoid wound, a subject which is at present one of the most interesting in otologic surgery. While the results of the socalled blood clot operation, as originally recommended by Dr. Blake, have not been entirely successful, and the reports of various surgeons have been so at variance, renewed interest in the subject was caused by the report of Dr. Holmes of his cases, at the last International Otological Congress held in Boston.

That a method of treatment of the mastoid wound which will more closely approximate the methods of the after-treatment of modern aseptic surgery in other parts of the body must appeal to all otologists is but natural. That it is also doubtless true that in a number of cases results can be surely obtained by primary closure, involving a principle of healing without drainage, must be assumed as absolutely certain, and the speaker himself has only recently operated on a patient with a nine weeks' acute staphylococcus purulent apex mastoiditis, in which after primary closure of the mastoid complete healing and drying of the mastoid wound occurred in sixteen days. In this case a drainage through the middle ear or the eustachian tube could be entirely eliminated, inasmuch as the apex cell was unusually large, invading the whole mastoid in a horizontal direction, and thus precluding all possibility of any drainage through the middle ear. We must therefore assume that in all cases of primary closure the gradual healing takes place by means of formation of organized granulation tissue.

Dr. Blackwell had laid special stress on the method of operation, as, for instance, in exposing the incus or almost completely removing the posterior wall. The speaker did not believe that the successful results of primary closure were due to such changes in the technic of operation, for in the cases in which he has operated with primary and secondary closures, the mastoid was opened thoroughly in the usual manner employed in the cases which had been treated by the open method. Nor is the method of primary closure, in his opinion, dependent on the filling up of the cavity with blood, although theoretically any method which closes a bone cavity that is not filled, necessarily involves the filling up of the cavity with some blood and is, in a way, a blood clot, or modified blood clot method. On the contrary, in the cases in which Dr. Cohn has so far employed this method with good results, he has, after thoroughly flushing the mastoid cavity with a 1/6000 bichlorid solution, followed by normal saline, closed the skin wound under compression, without allowing the cavity to fill up with blood, making it a point to squeeze out as much of the sanguinary contents as possible, and then employing a compression bandage. A small cigarette drain is inserted and allowed to remain for two or three days.

Whether the method of closure should be employed in cases of very severe and extensive osteitis, in cases of perisinus abscess, or those of extensive exposure to the sinus or dura during operation, or in cases of mastoiditis due to measles, scarlet fever, etc., must still be left to future experience and to the judgment of the operator. Dr. Cohn stated that while so far he has not had the courage to apply the method extensively, after the experience reported by Dr. Blackwell, it was his opinion that the method of primary closure should be given greater scope, but with the reserve of informing all patients before operation that it might become necessary to reopen the wound and continue the treatment by the open method.

In cases of primary closure of chronic suppurative conditions, the argument has been advanced that in those cases we are not dealing entirely with primary closure, inasmuch as drainage is established through the auditory canal, and that those cases must, therefore, be eliminated from any statistics dealing with primary closure of the mastoid opening. It is possible that in a number of the acute cases operated upon by

Dr. Blackwell the question of some drainage through the canal or eustachian tube may have occurred.

Dr. Ard said that he thought the method was more generally used than Dr. Blackwell anticipated before presenting the cases. For the last two years he had been using this method in Plainfield almost entirely—in at least forty cases, with the exception that in only a few cases was the granulation tissue removed from the attic. He follows the method described by Dr. Blackwell with the exception of going into the attic, and does not take down so much of the posterior wall and sews up all cases to the lower angle of the wall. The result is that after recovery there is a very much better canal—the cases also recover much sooner. Many cases have recovered in a little over two weeks—two of them in twelve days. Contrary to Dr. Blackwell's experience, some of the best results secured by Dr. Ard were with children—the dressings are much easier and the method is much more satisfactory in every way for both the patient and the physician.

He has had two cases of sinus thrombosis develop, but did not think the method was in any way responsible, as the process was probably going on at the time the mastoid operation was done. The method is far superior to the older method of open wound and packing. Possibly it is not suitable for all cases, but he had applied it in his cases, and the longest period of recovery was two and a half months, and only two of those. No secondary operation had been necessary during the past two years, and all recovered.

Dr. Kopetzky said that he presumed that Dr. Blackwell was operating upon acute cases—the report as read failed to specify; and if he was correct in this supposition, then there are certain points to be taken into consideration from the pathologic standpoint that were not considered when the method of Dr. Blackwell is critically examined. No issue could be raised with the brilliant results shown at the meeting, but the question as to the nature of the cases operated upon must be questioned, for that has an undoubted bearing, not only on the method advocated, but also on the pathology of acute mastoiditis as it is generally understood at this day. One is forced to the conclusion that there is either something wrong in the view of the pathology of acute mastoiditis as we know it, or we must concede these cases to be of exceptional type.

In restating what all present probably know, it is to be remembered that the eustachian tube, the aditus ad antrum, and the antrum mastoideum are one continuous tubelike passage lined with mucous membrane. The antrum mastoideum is the backward projection of this passage. When the middle ear and the mastoid process becomes the seat of an acute infection, this whole passage is filled with purulent exudate, and the mucous membrane lining the mastoid antrum becomes very much swollen and thickened, and assumes an appearance of granular outline, very often mistaken for granulation tissue. Why continue the evisceration of this swollen tissue as far forward as Dr. Blackwell advocates? Why stop where he indicates? The remainder of the mucous membrane lining the rest of the tubal passage that was just referred to cannot be removed, and when in the usual procedure the mastoid process is cleansed of its purulent contents, all know that the mucous membrane returns to the normal; that is, the lining of the tympanic orifice of the eustachian tube and the lining membrane of the tympanic cavity become normal after the usual operation. Then why go so far forward in a painstaking effort to remove only a part of the general mucous lining of this region. No one heretofore has shown that it is the suppurating mucous membrane that is the essential factor demanding removal to effect a cure of an acute mastoiditis. Usually when this membrane is left in situ, and in the course of events recovery takes place, this mucous membrane becomes thinned and returns to the normal, just as the remainder of it must do, even under the technic advocated tonight, and then there exists less chances of subsequent recurrences, for men who have studied this question from the histologic side, have shown that when the membrane is removed under the mistaken idea that this constitutes one of the factors of the mastoiditis demanding removal, that there then is deposited in its stead, connective tissue, which often forms a funnellike opening into the granulation tissue mass, which finally fills up the excavation, and this funnel leads the slightest accumulation of mucus from the middle car into the granulation tissue in the excavated mastoid process, and is one of the prime factors in the formation of recurrences of secondary infections in this partially organized tissue. This is particularly true when the subject of acute mastoiditis in childhood and infancy is thought of.

Finally, the operation as described tonight endangers the ossicles and their suspensory ligaments, and there seems no reason to assume the risk of impairing their functional activity. When the operation in the cases of acute mastoiditis is limited to the removal of the purulent detritus and all the accessible infected cells of the mastoid process, and the mucous membrane is relieved of the presence of the irritating masses of infected material surrounding and lying upon it, then no such risks are entailed, and, in addition, the return to health of the parts also means a return to conditions as nearly as possible to those in which they were prior to the onset of the infection; and this means much toward finally establishing normal hearing. Dr. Kopetzky, therefore, could not see the rationale of the procedure, and asked Dr. Blackwell to state what he considered the rationale of it to be.

Speaking of the bloodclot aftertreatment, he conceded that some men had had exceptional results, especially when they employed the method of technic devised by Christian Holmes, Reik, Sprague and Blake. He stated that he had tried to repeat the results that these men had obtained, having operated and treated a series of cases in a similar manner at the Manhattan Eye, Ear and Throat Hospital, but he could only say that his own results were uniformly unsuccessful, the majority of the cases either immediately breaking down, or soon coming to secondary operation.

Dr. Held said that he understood that Dr. Blackwell's idea was to hasten the process of healing and to avoid a posterior depression. Dr. Blackwell resorts to the primary bloodclot, and in addition chisels away a portion of the external attic wall. In reference to the primary bloodclot, Dr. Held opposes this method for several reasons, as follows: the fear of closing up an abscess cavity with bloodclot when it is practically impossible to render aseptic a wound so thoroughly septic; that we have in the closed cavity ideal conditions for the growth of bacteria and the spread of infection: heat, moisture, a good culture medium, and the bacteria present; that there might be inclosed in the cavity microscopic particles of infected material; that with a virulent infection and a suppurating middle ear and mastoid cavity, more drainage for a reasonable time should be had than from the incision in the middle ear alone, and that the posterior wound creates a counter opening.

As to the chiseling away and curetting of the external attic wall, Dr. Blackwell advocates going in between the superior and external ligaments of the malleus. Working in such a confined and small space, even with the best of technic, would very frequently result in damage to the ossicular chain. Dr. Held cannot reason the necessity for attacking the external attic wall in a case of acute mastoiditis.

He recognizes the advantages that would be gained by any form of aftertreatment which would shorten the long period of convalescence, and has been using the secondary bloodclot in acute mastoid cases, but only after the suppuration in the tympanic cavity had practically ceased. In this method the objections enumerated before to the primary bloodclot have been overcome. He believes that it is a very risky procedure to risk a patient's subsequent hearing for the sake of shortening the aftertreatment, when such aftertreatment can be shortened without any risks either to the patient's hearing or to his welfare.

Dr. Seymour Oppenheimer said that there evidently was no general agreement as to the preferred type of operation, and that Dr. Kopetzky had struck a keynote in asking for the reason of the recurrences. There is a question still as to whether the procedure of vigorously curetting the antrum is not the responsible factor for a number of recurrences, by causing an alteration of certain histologic structures, predisposing to subsequent infection.

Dr. M. D. Lederman said that the cases presented by Dr. Blackwell showed the excellent results he claimed. At the last meeting of the International Congress on Otology at Boston, the question of leaving undisturbed the tissues of the middle ear was discussed. Mr. Heath, of London, had emphasized this point.

For the last two years he himself had been following the technic suggested by Dr. Blackwell, without, however, curetting the granulations in the middle ear. The entire wound is closed, a cigarette drain being introduced in the lower angle, which is removed after the third or fourth day. The cases have all acted very nicely. The return of the hearing and the reformation of the membrane had exceeded his expectation. If that much can be accomplished through curettage of the mastoid cells and by simple drainage, without invading the middle ear, we should avoid the danger of disturbing the ossicular chain. He had not removed more than half the posterior wall of the bony meatus after removing the diseased cells; this was done for the purpose of allowing the canal wall to fall backwards. He was not inclined to go further than simple drainage. He inquired whether Dr. Blackwell attempts to cleanse out the cavity at his first dressing, or introduces another drainage tube. Another factor was the treatment of the wound at the time of operation. One is apt to use antiseptics which may interfere with the activity of the tissue. He was accustomed to use only a simple saline solution as a cleanser, and that may be an element in the healing of the wound, this permitting the tissue to return to its normal function without disturbing it by too heroic methods.

Dr. Kerrison said that he had been very much interested in the paper and in the discussion. He would not attempt to discuss the technic described by Dr. Blackwell, as he had not the personal knowledge of it which such a discussion calls for. It seemed to him, however, that such a presentation of the subject should be considered from the clinical standpoint, i. e., in the light of the reported results. The paper had in the first place the merit of honesty. Dr. Blackwell had not claimed uniformly brilliant results. He had stated that in some cases the period of postoperative repair or healing had been by no means unusual. He had also warned against the danger of a collapsed canal, if the surgeon neglected to pack the auditory canal firmly—a danger which would seem to be greater in this than in the older method of operating.

Dr. Kerrison said that as a colleague of Dr. Blackwell in the Manhattan Eye and Ear Hospital, he had been able to see some of the results of the cases reported very shortly after they were operated upon, and had been impressed with the rapid repair both of the drum membrane and of the post-auricular wound. As a very straightforward report, based on actual experience, he regarded the paper as a valuable contribution to the subject.

Dr. Blackwell, referring to what Dr. Cohn had said about drainage from the canal, even when the first dressing was removed the canal packing was usually absolutely dry. Occasionally there was some moisture, but the second dressing nearly always showed that there was very little infection remaining in the middle ear after the operation. Sometimes the canal drained for two or three days, or perhaps a week; but it was usually dry at the first or second dressing. In one of the O. M. P. C. cases there is still some canal drainage.

As to the cases referred to by Dr. Ard, in which the mucous membrane grows out of the middle ear into the mastoid, he had seen such cases and believed that it happened because after a thorough operation the cases had been packed tightly, especially the antrum. If the attic is curetted and the packing omitted, this complication will not occur. He believed that if any of these cases had been packed tightly in the posterior wound the healing would have been greatly delayed, beyond even the normal time.

Dr. Kopetzky had inquired concerning the rationale of taking out the antrum and attic tissue in acute cases. The paper is a purely clinical one, and the tissue had been removed for no other reason than that the subsequent results were good. That seemed a sufficient reason. When the infection is removed so far as possible from the mastoid and attic, the best that can be accomplished for the patients has been secured.

Dr. Held had spoken of the danger of closing an abscess cavity. From a macroscopic standpoint, at the conclusion of the operation these cavities were absolutely clean. We cannot remove microscopic evidences of the disease, but it would seem from these cases that the patient could take care of what remained. That was what was discovered in doing the work. He had not had any recurrences where the attic had been cleaned out.

Demonstration of the Strobilion, an Apparatus for Controlling the Voices of Singers and for Teaching the Deaf to Sing.

Dr. E. W. Scripture demonstrated an apparatus which he had invented and called the "Strobilion," which is used for controlling the voices of singers and for teaching the deaf to sing. He said that the idea was derived from an apparatus of his, described in 1896 in the Studies from the Yale Psychological Laboratory. A disc, fixed on the axle of an electric motor whose speed can be regulated by a rheostat, is divided into fifteen rings of alternating black and white spaces. The number of black spaces in each ring corresponds to the relations of vibrations in the diatonic scale. The inner ring has eight spaces; the next, nine; and so on. When the motor is going, the disc revolves and its entire surface appears an even gray. In front of the disc there is a scale marked with the syllables do, re, mi, etc. The disc is illuminated by a small flame from a flame box supplied by acetylene. When a tone is sung into the mouthpiece of the flame box, the flame vibrates and the disc is illuminated as many times a second as there are vibrations in the tone sung. Between these illuminations there is darkness. If the black spaces of any ring of the disc are passing a fixed point at a rate equal to the number of vibrations of the tone, that ring will split up into a series of black and white spaces that appears to stand still. All of the other rings will remain gray.

The deaf pupil is required to produce a tone that will make a certain one of the rings stand still. If the pupil sings a tone that nearly hits, but not exactly corresponds to a certain ring, that ring will split up into spaces, but instead of standing still it will appear to revolve forward or backward. If the tone sung is too sharp, it travels backward; if too flat, then forward. If the error is small, it travels slowly; if large, then rapidly. In this manner the deaf pupil can be taught all the notes of two octaves.

After the deaf mute has learned to sing, he can readily be taught to modulate his voice musically in speaking. The harsh monotonous voices of the deaf mutes are replaced by pleasant ones. Since each minute variation of pitch is rendered visible to the eye, the strobilion is also used for training singers to regulate the pitch of their voices accurately.

DISCUSSION.

Dr. Harris Taylor (principal of the New York Institution for the Improved Instruction of Deaf Mutes and Others) said teachers of the deaf were entirely unlike physicians, inasmuch as when the latter were told that any one had found a cure for any disease, his word was accepted at once (??), whereas the teacher of the deaf, when told that some one had found a means of controlling pitch in a deaf child, did not take it very seriously, but required to have it demonstrated. Quite a while ago, Dr. Scripture had written to him that he had found a means whereby a deaf child could be taught to control pitch. He had seen others who had exhibited limited enthusiasm on the subject and were interested, but did not believe that it could be done. What had just been demonstrated was evidence that a person can see whether or not his voice is going high or low. At first he had seen this work theoretically, and it seemed that it ought to work. The next thing was to try it with deaf pupils. He had sent to Dr. Scripture the worst deaf children that he had, on the theory that it could not do them any harm. Nearly all of them were cases of offensively high pitch, and no success had been secured toward improving them, and yet the results from the aid of this instrument were very gratifying indeed. One girl who had an abnormally high pitch had been with them for four or five years, and no change had been made in her pitch; her voice was almost a screech, yet with this instrument Dr. Scripture had been able to guide her voice up and down and to maintain such a pitch within her compass as he desired. She had been under Dr. Scripture's training for only a very short time, and she is now speaking several tones lower in her ordinary speech. This is a very important feature in teaching the deaf to speak. Very often their speech is very offensive; a great robust boy has a voice which goes up and down, and a nice little girl may have a deep, low voice. It jars on one's sensibilities, even if one understands what is said. Dr. Scripture is doing a great deal for the children who have been sent to him. The influence is noticed in their speech even after one week, and the progress is noticeable. It can be demonstrated by watching them. Some of them can be taught to control pitch a great deal better than some normal persons. Dr. Taylor said that he could not speak too enthusiastically of the influence which this apparatus can have on the voices of the deaf, making them more natural and more easy to understand, as well as more musical in their speech.

Dr. W. Hallock (Columbia University): I wish to express my appreciation of the opportunity of being here. I am only troubled with the thought that if I should undertake to mention all the things this apparatus could be used for, many of you would want to go home before I got through. Like many other discoveries, it seems wonderful that it has not been thought of before. It is a simple and ingenious method of doing what we have been trying to do in such complicated ways. If you go to a moving picture show you may note that in a scene which shows wheels with spokes the exposure of the shutters of the kinetescope is not the same as the movement of the spokes of the wheel, and the wheels appear to turn slowly either way. If the rates are the same the wheel will appear stationary. One day I got all ready for a terrible accident on the railroad, for I saw the wheels of the rapidly moving truck were standing still; but nothing happened; the train went on smoothly—and then I recognized that I was looking through a picket fence.

I have had a good deal of experience one way and another with these things. I was fortunate enough a good many years ago to see and hear a patient who had had his whole larynx taken out and could talk perfectly well, but in an absolute monotone. Then I was very much interested in the control of pitch in the deaf, for some years ago I had occasion to make some experiments with Miss Keller, who carries her pitch very well by putting her fingers on the control larynx. As long as she has her fingers on that she does very well, but if the control stops, she loses her control.

The possibilities of this instrument are way beyond what Dr. Scripture has suggested. The whole community ought to raise a monument to any one who will do anything to keep singers on the pitch. They cannot tell half of the time by their own ears whether or not they are on the pitch. This will give them a means of doing that.

Another thing that it is available for and very valuable: many singers sing and you cannot tell whether or not they are on the pitch, for the pitch tone is almost lost in the high notes and overtones. Suppose, for instance, you take off the present disc of two octaves and put on the fundamental and first seven overtones; you can change the fundamental by changing the range. A man sings bass C; in most of them you will get very little bass C. Many times you get the overtones, and the person who sings ought to be able to get the fundamental.

Some of them, I know, are anxious to have some way of finding out whether they get the fundamental or not. They can tell themselves with this device. It seems to me that it is going to be very valuable in that way. Of course, a person has to have some practice, and you must look out for enhanced resonance, but that is easily overcome. Possibly the uncertainty in the tremolo or the inability to hold the pitch steady is due to our own inability to give an even pressure of air, either in our own voice or blowing in the pipe. It is almost impossible to blow with a perfectly uniform pressure, and if you change the pressure you change the pitch in almost any wind instrument. I tried to notice when the experiments were being made whether it had anything to do with the pulse beat. If you watch the wheels of an automobile, the wheels will seem to stop for an instant. Count your pulse, and you will find that the stop is every pulse beat. When the blood is forced into the retina there is a momentary blur.

It must be a very valuable thing to the deaf and to the teachers of the deaf, for it is right there. There are no reductions or calculations. The person sees the whole thing. I am very enthusiastic about it, and congratulate Dr. Scripture most sincerely.

An Improved Method of Eliciting Rotation After-Nystagmus.

Dr. Edmund Prince Fowler: It is generally accepted as a fact that because of the manifest impossibility of rotating one labyrinth without its fellow, it is also a fact that it is impossible to separately test by rotation one labyrinth unless the opposite be diseased or dead, and even in these cases conclusions can be definite only in certain cases.

I will endeavor to convince you that by my proposed method of rotation it is possible, both theoretically and practically, to separately test the labyrinths of either side, whether one or both be in health or disease. I realize the intricacies and difficulties of labyrinth physiology and pathology and the many arguments which can be brought forward to combat my ideas. Partly for that reason, partly because I have not had sufficient experience with this new method to state definitely some of my observations, and mainly because I believe it will be better to first present my simple arguments, I bring forward at this time the tests in relation only to the horizontal semicircular canals. However, these being true, it is self-evident that it is possible to apply the principles to each and every canal in the two labyrinths.

I will state my basic facts and arguments in as simple words as I am capable of using, that those not familiar with the hydromechanics of the canal systems may readily follow my brief argument.

If both normal horizontal semicircular canals (referred to herein simply as canals) are revolved in a horizontal plane, and about a center equidistant from each, the effects of equal rotations to the right or to the left will be equal; the nearer the common center of rotation is approached, the greater become the effects following rotation; and the farther it is removed from the canals, the less are the effects.

One canal being destroyed, the remaining canal will be affected more or less, depending upon its nearness to, or distance from the center of rotation; and if the axis of this center be within the circumference of the canal, the greatest possible effect will be obtained.

If two canals are placed on the same radius of rotation, the centripetal will be nearer the common center of rotation by a distance equal to its distance from the centrifugally placed canal; one canal will, therefore, be subjected to greater effects following rotation than its mate.

The nearer to the center the inner canal is placed, the greater will be the differences between the effects upon the two canals during and following rotations.

If both canals are equally normal, it is obvious that the results following rotations to the right or to the left will be equal, unless the direction of the fluid flow makes for a difference—which I may here state it tends to do; but I will not go into this factor at this time, as it complicates, though aiding, my argument for the use of tests here brought forth.

The results will be practically equal also whether the right or the left canal is centripetally or centrifugally placed, if they are accurately so placed and maintained.

If one canal is diseased, what happens? The results will differ, not only (in certain stages of labyrinthitis) as between right and left rotations, but also as between central and distal placement of the functionating canal. For instance, let us assume that the left canal is dead. Following the usual method of rotation, we may, or we may not, obtain sufficient differences in the duration of the nystagmus following right and left rotation to enable us to make a diagnosis. Following right and left rotation, with the radius of rotation passing through the two canals, may likewise lead to doubtful results, following rotation, say, with the left canal peripheral; but turn the head on its own axis through 180 degrees, and thus bring the right canal peripheral (or move the head laterally to the right of the center of rotation); the right canal will now be in a position further from the center by approximately 80 mm., and though the differences in the nystagmus between right and left rotations be unsatisfactory, yet the duration of these eye movements will be less than those obtained when the healthy right labyrinth was tested nearer to the rotation center. By first rotating with both canals at given and equal distances from the axis of rotation, and then placing one canal at this same position and the other farther out on the common radius of rotation, it is apparent that we have a means of testing both canals, first together, and then separately.

It so happens that the labyrinth is about one and a half inches from the side of the head about the external auditory meatus, and also one and a half inches from a point midway between the two labyrinths. If, therefore, we rest the head against a support placed about the ear and on the axis of rotation of a revolving chair, one of the two labyrinths will be placed one and one-half inches, and the other four and one-half inches from the axis of rotation.

If we fix the head between two supports, one of which surrounds each pinna, so that the midpoint between the labyrinths coincides with the axis of rotation, both canals will be about one and a half inches from the axis of rotation.

Hence, in the lateral positions, one canal—and only one—will be one and one-half inches distant from the common axis of rotation, or exactly in the same position relative to the axis as it was during the position with the axis midway between the two canals.

Of course, the position of the ampulla of the centripetally placed canal is changed relatively—being in the first instance centripetally, and in the second instance centrifugally placed in relation to its canal.

There is one drawback to my method, namely, if I rotate ten times in twenty seconds, I may cause excessive stimulation of the centers and bring on nausea and vomiting. But I find it is not necessary to rotate ten times in twenty seconds. In fact, fewer rotations give more accurate results, mainly because they occasion less nervous excitement and less shock to the canals.

A little later I will go into this subject more at length and submit the technical calculations which have been made at my request by competent engineers. These prove mathematically what I have proved experimentally and clinically.

The chairs now used for the rotation tests are so constructed that the labyrinths take a position five or six inches from the rotation center. This is one reason for bad results, as any movement of the patient's head varies the distance of each labyrinth from the center of rotation a double amount, because if one side moves backward and away from, the other moves forward and towards the center of rotation.

DISCUSSION.

Dr. Hallock said that he did not know anything about nystagmus, but was very much interested in the presentation of the subject, as he has occasion to lecture about the acoustics of the ear. The semicircular canals are pretty near by, and some one is pretty sure to ask what they are for. He wished to add his opinion as that of an hydraulic engineer to say that the mechanics of the case as Dr. Fowler had presented them were entirely correct.

Dr. Friesner said that the method presented by Dr. Fowler was exceedingly ingenious, as were most of the things which he invented. When, for purposes of comparison, we apply any test to both labyrinths, or attempt to apply the same mechanical force to both labyrinths, we must take the utmost pains to see that the irritation is applied equally and simultaneously. In the old method of rotating for testing the external or horizontal semicircular canals, we so fix the head that both labyrinths bear such relation to the axis of rotation that the mechanical force engendered acts upon them both equally and simultaneously. One serious objection that this method seems to present is that in shifting the head we may not apply the same force to both labyrinths and, therefore, even though we do get differences in the after-nystagmus, these differences may be due at times to errors that slip in through misplacing the head, through inaccurately placing the two labyrinths with relation to the axis of rotation.

Dr. Fowler said he would show that what Dr. Friesner called the older method, but which he would refer to as the present method of rotation, was at a mechanical disadvantage and therefore less accurate than the method introduced this evening. In the author's method, the head is so placed that the two canals are resting on the same radius of rotation, but in this older way, the accepted way, the canals are on separate or opposite radii of rotation. As a matter of fact, in all the chairs that he had examined, the head rest is eight or nine inches back of the center of rotation. Now what happens? The slightest change of one labyrinth backward or forward makes the same amount of change in the opposite labyrinth, but in the opposite direction, and hence the error is doubled; whereas, if the head is placed with the labyrinths on the same radius, even considerable movements make no appreciable difference in the distances the labyrinths are from the axis of rotation, and consequently only very slightly affects the duration of the after-nystagmus. It is readily seen that the proposed method of rotation may shed much light upon several interesting points in labyrinth physiology and pathology. A fortunate circumstance is the fact that his tests depend not wholly upon the accuracy of his reasoning, but simply upon the acknowledged fact that the effects of rotating any body vary with its distance from the axis of rotation.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of December 17, 1912.

THE PRESIDENT, DR. JOSEPH C. BECK, PRESIDING.

PRESENTATION OF CASES.

DR. CHARLES H. LONG reported a case of

Atresia of the External Auditory Meatus Due to Furunculosis,

of which a detailed report will be given at a subsequent meeting.

Nasal Tuberculosis—Recovery.

DR. OTTO T. FREER reported the case of a woman, aged 59, first seen on May 28, 1912. At that time the affection had existed for five years. The symptoms consisted of nasal stoppage, with complete obstruction of the right nostril and partial closure of the left. Scabs formed upon the septum and were cast off and reproduced. In 1910, as the result of an examination of microtome sections in the laboratory of the University of Virginia, the condition was declared nasal tuberculosis. The nose was then repeatedly cauterized and the disease seemed checked, until the symptoms returned in March, 1911. The diseased area was again cauterized with nitric acid and the galvanocautery, and a perforation formed in the septum.

Examination of the patient showed exuberant granulations upon both sides of the cartilaginous septum, these granulations nearly filling a perforation which lay about one-half inch above the nasal floor and in a vertical line with the anterior nasal spine. The anterior inferior part of the septal cartilage was softened so that the nasal tip could be readily depressed with the finger. Following the alar cartilage, granulations lined the external nostrils. There was a granulating area which covered the anterior end of the right inferior turbinate and an area of the septum opposite it.

On account of the involvement of the external nose, the condition seemed inoperable without great disfigurement, so the patient was referred to Dr. Adolph Hartung for X-ray treatment twice a week. A specimen was also excised from the border of the perforation and declared by Dr. Maximilian Herzog to be tubercular. Under the influence of the X-ray the granulations disappeared from the external nares and from the perforation in the septum, and the softening of the cartilage ceased to progress. The granulating areas upon the inferior turbinate and the septum further back were not influenced, however, because of their depth in the nose, so that on June 8, 1912, the right inferior turbinated body, which was tubercular throughout and was softened into a mass of spongy granulations, was extirpated, while the area opposite it upon the septum was cut away to the bare cartilage and bone. Healing took place rapidly, and at the present day, December 23d, all evidence of disease has disappeared from the nose.

The history of this patient shows that an inoperable case of nasal tuberculosis may be brought into a condition where operation is possible by the use of the X-ray. Its effect in this case confirms the experimental findings of Professor Brünings, of Jena, who proved, after producing inoculation tuberculosis in the larynges of animals, that the X-ray is more effective in the treatment of local tuberculosis than intense light.

Dr. Freer also referred to a case of nasal tuberculosis in which he had extirpated the entire ethmoid labyrinth upon the right side, the right middle turbinate, a large portion of the covering of the septum upon the right side, and a tubercular tumor from the posterior border of the vomer in 1907, the patient making a complete recovery. (This case was reported in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, March, 1910.)

Total Resection of Superior Maxilla for Carcinoma.

DR. JOSEPH C. BECK presented a fresh specimen of a case of total resection of the superior maxilla for carcinoma, beginning several months ago as an epulis. The patient was a man, 44 years old, otherwise in a healthy condition; there were no glands in the neck as yet infected with cancer. Preliminary exposure and compression of the external carotid artery was performed. The typical Langenbeck operation, with few modifications, as curettement of the ethmoid cells, was carried out. The anesthetic was ether by vapor, carried down into the larynx. There was very little bleeding present. A large defect of the cheek remained, owing to carcinomatous involvement of this structure. The patient was returned to bed in good condition. The gross specimen shows clearly on section the presence of carcinoma, and a previously excised piece on microscopic examination also proves it to be an alveolar carcinoma.

The point Dr. Beck wished to make was that all these carcinomata should be operated upon, especially so long as a patient is in good general condition, and especially if the glands in the region are not metastatically involved, and if they are, they should be removed en masse. The mortality is great, recurrences frequently occurring, but if only one case in every twenty-five is saved, it may be said to be a percentage of one hundred, since without operation they all die.

Technic and After-Treatment of Resection of the Septum Without Packing.

Louis Ostrom, M. D.,

Rock Island.

The author prefers to have the patient sitting up during the operation. There is less bleeding, and the patient can assist in blowing his nose, etc. He is less fatigued with the patient upright than when operating with the patient in the recumbent position, and the position seems more natural. The average time required for the operation is forty-five minutes, including preparative operation and immediate after-care. Many cases have been completed in fifteen minutes; some few cases have taken two hours.

Time of Operation.—Usually between seven and nine a. m., so that patient may have a full meal in his stomach; the general effect of the cocain is less disagreeable, and if anything should arise demanding our attention, all day is at our disposal.

Anesthesia.—Flake cocain is used alone, applied three or four times in small quantities, until the tolerance of the patient becomes manifest. When the patient is fully anesthetized and not sick from the cocain, and everything ready for the operation, adrenalin is used. In all his experience he has had practically no bleeding during the greater part of the operation, unless adrenalin was used too early. It takes a little longer to get started, but saves time in the end.

Technic.—The primary incision is made about vertical, one-third to one-half inch long, pointing down toward the middle of the incisor crest. This primary incision in the mucous membrane is made at right angles to the surface. The elevation of the mucous membrane is made by first scraping or pushing the perichondrium from the cartilage, so that the cartilage is distinctly visible within the primary incision. The mucous membrane is then elevated backwards above the vomerian crest and up to the upper border of the deflection. With his right-angled elevator (made for him by Ermold, of New York, in 1903) he elevates the mucous membrane up to the roof of the nose below the bridge, and forward to the primary incision. The primary incision is then extended upwards to near the roof of the nose, then down to the incisor crest and out about one-eighth inch across the floor of the nose down to the bone (Yankauer). The periosteum above, below and over Jacobson's cartilage is separated from the bone and cartilage with Stevens' dull pointed tenotomy scissors bent on the flat, the points of which hug the bone and cartilage and cut the fibrous tissue connecting the periosteum to the sutures at the incisor crest in the region of Jacobson's cartilage. This dissection with Stevens' scissors is continued backwards to the posterior end of the incisor crest, when a dull, narrow elevator can elevate the mucous membrane on the floor of the nose adjacent to the septum, then upwards on the septum up to angle of deflection on to vomerian crest. The mucous membrane over the crest or ridge, or spine, is then elevated from behind forwards, or this process is left until the bony septum is cut above and below. The edge of a sharp ridge can be cut off with Stevens' scissors and left adherent to the mucous membrane, thus avoiding tedious dissection and possible perforation.

The incision in the cartilage is about one-sixteenth inch back of the incision in the mucous membrane, but in the same general direction, but at an angle of forty-five degrees to the plane of the septum, and only about one-quarter to one-third inch long. One finger is placed in the opposite nostril behind the incision, and pushes on deflection so that the incision in the cartilage gapes.

The point of a blunt-pointed elevator with the tip bent on the flat is pushed into the incision of the cartilage, and while pushing against the cartilage anterior to the incision with the bend or heel of the elevator the tip is directed against or toward the cartilage of the septum (never against or toward the mucous membrane), and as soon as the tip of the elevator disappears from view, the finger in the concave nostril feels the elevation. At this point ocular inspection must take place, to be sure that the septum was not perforated, cut or torn. The elevator is then pushed backward, elevating the mucous membrane backwards, up and down as much as it can. The author's right-angled elevator is then used to elevate the mucous membrane from behind forwards, first above, then below, down to the floor of the nose. The incision in the cartilage is then prolonged upward to one-quarter inch from the bridge of the nose, and downwards to the crest. Remembering that the fibers connecting the vomerian sutures to the periosteum and perichondrium run out and back, by pulling forward these fibers guide the instrument in against the bone, and no matter how scarred or adherent or grooved the concavity of the septum may be, the right-angled elevator separates the mucous membrane from the bone and sutures, away down to the floor of the nose, as far forwards as the anterior end of the incisor crest, with no likelihood of perforation.

Ballenger's swivel knife is used to cut the cartilage above, accurately guiding it to cut upwards parallel to the bridge of the nose, one-third inch from the upper border of cartilage of septum or bridge of nose. The rest of the cartilage is removed in one sweep. The bony septum is removed with Killian, Grünwald, or any other forceps or punches which one may prefer. The maxillary crest is removed with gouge, chisel or punches.

Usually two black silk sutures are placed in the mucous membrane, one in the middle and one at the angle on the floor. A plug of cotton dipped in paraffin-vaselin is placed over the incision. This can be removed and renewed as desired.

CONCLUSIONS.

1. Hematoma does not occur if there is free drainage, and all blood will escape from septum in two to five days, usually after the third day.

2. In case of infection, drainage is already prepared for. He has had no infection of the septum.

3. Hemorrhage cannot take place unless the mucous membrane is so mutilated that an open wound is produced.

4. Packing cannot be placed uniformly on the septum; usually the upper one-half or one-third is never packed. (His observation at all clinics, and numerous operators.)

5. Pain follows removal of packing.

6 Hemorrhage may take place when packing is removed, compelling repacking.

7. General sinus infection and severe pain usually follow packing.

8. Mucous membrane of turbinates may be injured by pressure from packing, and especially on its removal.

9. Hematoma often occurs after removal of packing.

DISCUSSION.

Dr. E. Fletcher Ingals is not in sympathy with removing so much of the septum as is usually done. If bone and cartilage were not needed, they would not have grown there in the beginning. The idea of removing as much of the bone as possible without having the nose fall in is a mistake. The less bone removed, the better for the patient. He had seen many patients on whom this had been done, but he could not recall any in whom there was a firm septum. He thinks that usually if the bent portion of the septum above the most prominent ridge of the spur is retained, it is much better for the patient. In very many cases this portion of the septum is nearly half an inch in width; it can be taken out much more easily than it can be retained, but he thinks it better for the patient to leave it. The same principle should apply to the cartilaginous septum. He thinks that usually much more of it has been removed than necessary.

Regarding the author's results from not packing, they seem to have been excellent. Dr. Ingals thinks that packing, in the majority of cases, is a necessary means of safety. If no opening is made through the mucous membrane on either side except the one directly in front, he has no doubt that clotting will usually, perhaps always, stop the hemorrhage, unless the patient happens to be a bleeder.

Dr. Ingals then stated that he wished to present to the society an efficient and easily removable nasal packing, for, notwithstanding the fact that many patients who have suffered operations in the nares might go to their homes without packing and escape serious hemorrhage, yet in many, bleeding that alarms the patient comes in spite of the packing. In a considerable number, bleeding that is alarming to the surgeon comes on, and in a few fatal hemorrhage has occurred; therefore, he believes that for many reasons careful packing of the naris should be made in most cases after cutting operations on the septum, turbinated bodies, or accessory sinuses. Sometimes packing is required after the removal of mucous polypi, but usually it is not necessary when only a snare has been employed. While in a large number of cases, without packing, no serious hemorrhage would occur after the usual operations, it cannot often be desirable to waste even two to four ounces of blood. General surgeons prevent all bleeding possible, and their reasons for doing so would largely be valid in intranasal surgery; therefore, he cannot think we are justified in subjecting patients to the risk of bleeding. Usually bleeding would not occur for several hours after the operation; then, if the patient had gone home, he would at least be likely to be greatly alarmed. It seems to him unfair to subject the patient to this distress, aside from the first injury resulting from the loss of blood. If there were no other reasons for packing, the peace of mind and comfort of the surgeon make it well worth while.

Often the parts are in such condition that packing is necessary to hold them properly in place.

He has known of several very alarming hemorrhages after intranasal operations, in which the patient's life was saved only by prolonged and persistent treatment, much of it very painful to the patient, and he has known of two or three fatal results. One of these occurred in his own practice, the patient dying from the effects of secondary hemorrhage, although he had stopped the flow thirty-six hours before this sad termination.

The only objections that he can see to packing of the nares after cutting operations are: (1) Obstruction of respiration; (2) pain from pressure; (3) prevention of the escape of pus, and (4) pain from removal.

The obstruction of respiration cannot be considered a real objection, because usually the patient has already become accustomed to this inconvenience, and even if packing were omitted, swelling of the tissues resulting from traumatism would usually stop the naris.

Pain from pressure is a serious objection, if the packing is too firm. The surgeon's experience must guide him to get just the right amount of pressure; and he should make provision against pain by suitable analgesics. He is accustomed to give the patient five or six doses of five grains each of phenacetin, directing one every hour for four or five hours, if required, for pain. At the end of this time, if the pain is still troublesome, or if the patient is nervous, as he is likely to be from the cocain, he gives him half a grain of codein, and has the dose repeated in two or three hours, if needed.

Purulent discharges after operations for infected sinuses will almost necessarily occur, and if the naris is firmly packed the pus may be forced back so as to cause infection of other parts. This danger cannot altogether be avoided, but it should be guarded against by removing the packing as soon as the danger of hemorrhage has passed. Usually the packing may safely be removed within from sixteen to twenty hours.

Pain caused by removal of the packing is by far the most serious objection to its use. To avoid this various methods have been adopted, most of which fall very short of the desired result, as can be appreciated only by those who have themselves suffered from the operation. The same remark would be applicable to the dressing after a large percentage of operations, and he feels that there is no one thing in which surgeons are so remiss as in their failure to prevent suffering at such times.

He has tried many different dressings after intranasal operations, designed to prevent secondary hemorrhage and avoid pain at the time of their removal. The most satisfactory one is made of rubber sponge, the form of which is peculiarly adapted to favor coagulation of blood. It is made of rubber sponge, preferably about 4 cm. thick, and from 5.5 cm. to 8 cm. long. In preparing the packing the sponge is grasped tightly and compressed between the blades of a long strong forceps, like Pean's hysterectomy forceps, ten inches long, or like the speaker's straight esophageal forceps. The blades are closed tightly as possible, and locked by the catch on the handles. Then the sponge is trimmed off with scissors close to the blades. When the blades are opened, the sponge at once expands, and we have a piece of rubber sponge which should have been cut to about 2 mm. greater in thickness than the extreme width of the naris we wish to pack while the tissues are shrunken down with cocain and epinephrin. The edges of this piece are trimmed down so that it will fill the naris vertically, and when there is fear of hemorrhage far back, the distal portion of the sponge should be left about 2.5 cm. longer, and about 5 to 8 mm. less in height (width). This is done so that this portion, after being pushed through the naris back to the pharyngeal wall, can be drawn forward to plug the choana. For this purpose the whole piece should be about 8 cm. long, whereas if only the naris requires protection, a piece 5.5 cm. in length will be long enough. It is important that this sponge be not too thick, otherwise, in spite of its softness, pain will be caused by the constant pressure that it will exert. The sponge having been fashioned to fit the naris, a strong linen thread is passed through from above downward, about 1.5 cm. back of its proximal end, and the two ends of this thread are tied together, so as to form a loop about 4 cm. long, by which the sponge may be withdrawn. To prevent the sponge from being torn apart in removal, a similar thread is sewn through and through from before backward at the upper and lower part of the packing, and this is tied in front of the thread passed vertically in forming the loop for extraction. Thus prepared, when the loop is pulled upon, all the sponge will have to come with it. When it is desired to plug the choana more carefully, the long piece of rubber is used. A strong thread is passed through the sponge about 4 cm. in front of its posterior end. This is then fastened to the posterior end near one edge, and sewed over and over along the posterior end to near the other edge, when the thread is carried forward and through the sponge at the same distance from the posterior end as the other end of the thread. The two ends of the thread are then tied together, forming a loop about 7 cm. long, which, when pulled upon, will double the back end of the sponge upon itself and draw it into the choana. When the packing has been made ready, it is rolled up, compressed tightly and immersed, and allowed to expand in a strong solution of formalin. While thus immersed it is alternately compressed and allowed to expand until the solution has permeated every part. It is then allowed to soak in the solution until fully sterilized and then is washed thoroughly in sterile water. It is then ready for use. In introducing this packing it is grasped firmly between the blades of a strong forceps shaped like the ordinary ear forceps, but the blades should be about four inches long, and there should be a catch to hold them together until the surgeon is ready to detach them. The mucous membrane is then smoothed down, and long thin spatulas introduced on both sides of the naris to prevent too much friction as the sponge is crowded in. With the forceps the packing is then pushed back as far as desired, the forceps are released, and the sponge expands to its full size. Something is then placed against the anterior end of the sponge to prevent it from being pulled forward, and the forceps and spatulas are withdrawn. It is usually best to push the sponge back somewhat farther than it is to be left, and later draw it forward to the desired position. A pledget of cotton is then placed in the nostril, and the patient may go home confident that there will be no serious bleeding. If it has been a case where the choana was to be specially packed, while the sponge is held back by something pressed against its anterior end, the longer loop is pulled upon, whereby the posterior end of the sponge is doubled on itself and drawn into the posterior naris.

After fifteen or twenty hours this packing may be withdrawn, often with no pain, and always with much less than caused by other packings that he has tried. When for any reason a large packing has been put in and traction upon the loops for removing it causes pain, the patient may be given a few whiffs of chloroform or some other anesthetic, and then the packing can be pulled out quickly. For this purpose he uses chloroform in the following manner: In the bottom of a two-ounce, wide-mouthed bottle he places a sponge that will about one-third fill it, and on this pours a dram of chloroform. The patient, holding this in his hand, places the mouth of the bottle between his lips and breathes deeply at the rate of about thirty respirations per minute, until he begins to feel the effects by dizziness or a sense of losing himself. Then the surgeon, who has hold of the loop, pulls the sponge out in about a second, without causing pain. The patient immediately recovers from the chloroform. There is no possible danger from using chloroform in this way, for the patient would drop the bottle before becoming unconscious or taking too great a dose. Holding the bottle in the hand in this way warms the chloroform and assists in its rapid vaporization.

In cases when for any reason he has had reason to fear hemorrhage, he has rubbed on those portions that would come in contact with the wound a powder consisting of quinia urea hydrochlorate, 1 part; acid gallic, 1 part, and acid tannic, 3 parts. This causes a clot firmly adherent to the mucous membrane, and also enmeshed in the rubber, so that withdrawal is apt to cause pain, unless some anesthetic is employed, but it may be pulled out quickly. He thinks that it is seldom necessary to use the astringent powder, and that the patient is more comfortable without it; nevertheless, in those predisposed to hemorrhage he would highly recommend it.

Dr. Otto C. Freer said that Dr. Ostrom's method of operating was simply the well-known Killian operation, with slight modifications. To enter the intricate and distorted anatomy of a septal deflection through a little cut, one-third of an inch long, as Dr. Ostrom did, was to make the submucous resection a blind operation in a field where minute vision is needed to avoid disaster. It is impossible, in deflections at all extensive, to do a thorough resection without an open operative field, and Dr. Freer had removed many bony deflections from patients for whom a submucous resection attempted previously by others had consisted of the cutting out of a piece of cartilage with the swivel knife through a buttonhole cut of the type advised by Dr. Ostrom, the operator, through this inadequate entrance, having been unable to take away the bony part of the deviation, which kept the nostril as much blocked as if nothing had been done.

Dr. Freer did not recognize in Dr. Ostrom's description of the anatomy of deflections the conditions he had encountered in his resections, and regarded the ignoring of anatomic facts of importance by Dr. Ostrom as proof that the blind mode of operation he had been following had not shown them to him. For instance, he made no reference to the frequent traumatic deflections with two or more separate fragments forming distinct cartilaginous plates of a sharp-angled deflection. A fibrous bridge passes from naris to naris between such fragments, and, by uniting the perichondrial covering of both sides of the septum, makes it impossible to pass the barrier formed by such a bridge without a sharp blade. Dr. Ostrom had advised the elevation of the coverings with a dull elevator moved from behind forward. This elevator would be arrested by the obstacles referred to, nor could it with safety be replaced by a sharp blade, if employed through a buttonhole cut. The dull elevator would also be ineffective in the many cases where inflammatory changes in the mucous membrane of the septum have left broad areas of intimate union between toughened cartilage and thickened perichondrium. These areas are never discovered until the denudation is under way, and sometimes include nearly all of both the convex and concave sides of the deflection. In such cases the denudation is one continuous dissection, and a dull elevator has no place. Dissection, however, requires an open operative field.

Dr. Ostrom does not mention the overlapping of the vomer by the cartilage in the vomerocartilaginous articulation, nor the crossing of the blended periosteum and perichondrium from naris to naris in this articulation, a condition repeatedly described by Dr. Freer, and one which makes dull denudation downward below this articulation impossible.

Dr. Ostrom speaks of the "anterior tip" of the septal cartilage and of the "anterior ridge." There are no such anatomic designations, and it is impossible to tell what Dr. Ostrom meant by them.

In regard to the removal of the bony part of the deflection, as described by Dr. Ostrom, Dr. Freer would have found himself quite helpless with the weak instrumentarium displayed by Dr. Ostrom in the face of the many massive deflections he had encountered. He had often taken away the anterior half of a deflected vomer half an inch thick, and vomeroethmoidal angles one-third of an inch in thickness. Such solid deflections have to be over- and undercut, as shown in Dr. Freer's last article on the submucous resection in the *Journal of the American Medical Association,* 1912. Unless these bony angles are severed from the perpendicular plate above with punch forceps, the attempt to break them out in the manner described by Dr. Ostrom is dangerous and liable to create fissures in the perpendicular plate which may extend through the sphenoidal sinus to the optic foramen, with resulting blindness in the eye involved, as happened in a case described by Professor Bernhard Fraenkel, of Berlin.

For denuding the incisor crest and anterior part of the vomer, the socalled "ridge," of its periosetum, the delicate scissors shown by Dr. Ostrom seemed peculiarly inappropriate, for these rough bony parts are covered with a periosteum so clinging that they can be shelled out of their covering only by the use of the raspatory designed by Dr. Freer, after the periosteum has been split to the bone with a strong knife. To do this, however, again implies an open operative field, for it cannot be done in the dark.

Dr. Ostrom attributed to the minute cartilaginous remnant of Jacobson's organ an anatomic importance it does not possess. It takes no part in the creation of deflections.

Dr. Ostrom's advice, not to pack after operations upon the septum, is not new. It has often been given in past years by those who like to startle by boldly contradicting the teachings of the experienced, and who advance such advice as progress, while in reality it is retrogressive discarding of what has been learned by the painful experience of the profession. The objection to such advice is that its authoritative tone is apt to mislead the beginners in the specialty into a disregard of common prudence. Why do rhinologists continue to pack, in spite of these innovators? Because neglect to insert the tampon in some case where it did not seem needed, because of absence of bleeding immediately after a submucous resection, was punished by a severe hemorrhage two hours after, when the contractile effect of the cocain and adrenalin upon the vessels had passed away and left them open and paretic. Packing had to be done then under most difficult conditions, with disturbance of the wound and the chance of perforating in a nose full of clots. The severe nosebleeds occurring spontaneously from little vessels upon the septum should show how dangerous it is to leave any cut vessel uncompressed in the nose until it has regained its contractility and is effectually closed. Where the buttonhole cut is used there is also danger of bleeding between the coverings of the deflection removed. Several patients operated upon by others through a buttonhole cut have come to Dr. Freer with large hematomas formed in this manner and blocking both nares. In two of these, abscess of the septum occurred. These hematomas could have been prevented by proper packing.

Dr. Freer's layer packing is made of strips of Johnson and Johnson's sterilized lint, cut into strips one-third of an inch wide, with sharp shears, and impregnated with powdered subnitrate of bismuth. The bismuth keeps the tampon aseptic and has been used by Dr. Freer for twelve years. In one case, a bleeding polyp of the septum, he left the tampon in for ten days without its becoming putrid. An iodoform tampon will begin to smell in twenty-four hours. To insert the tampon, the nostril is held open with two of Dr. Freer's septum retractors, one, a long one, being used to guard the site of the operation. A strip is then folded on itself in the middle, stretched taut upon a long dull elevator, and carried back to the posterior naris, when it is tamped down upon the nasal floor. A second strip is inserted in the same manner and placed upon the first. In this way layer after layer of parallel strips are laid in the naris until it is filled to the top. This tampon completely closes the choana by filling the space between the nasal floor and body of the sphenoid bone; it is elastic, and applies itself evenly, without unequal pressure, to all inequalities of the nasal interior. It causes no distress and keeps the flaps in place, and it gives absolute assurance that hemorrhage of moment cannot occur.

The tampon is removed in part in twenty-four hours, those strips being taken out which are in contact with the outer nasal wall or other strips. The strips immediately applied to the wound are left in for a day or two longer, until they have become nonadherent. As the tampon stays perfectly pure, there is no harm in leaving it, especially as the nostril should not be used for breathing for a week at any rate.

No clamp, such as has been advocated of late for the submucous resection to take the place of the tampon, can give the even, reliable pressure of the latter. Clamps are apt to slip, to press too much or too little, and are about as treacherous as the clamp used to control tonsillar hemorrhage, in respect to assuring a continuous stoppage of bleeding. A patient with a good tampon after a submucous resection does not need to stay in a hospital, but may be sent home—a great advantage, for most of them dislike the idea of a hospital. To leave a patient unpacked in a hospital, with the chance of an interne, inexperienced in nose work, having to pack the furiously bleeding nose of an excited patient, does not seem wisdom to Dr. Freer, as perforations and injury to the wounded parts are liable to occur.

Dr. Derrick T. Vail, of Cincinnati, said that when the submucous operation for removing the cartilaginous septum was first described he came to Chicago in order to see it done and to study the indications for the operation. In his work previous to that time he had never found cases that seemed to require the removal of the cartilage. The most that he did was an Ashe, a Row or a Gleason operation, but after his visit to Chicago he had entirely different ideas regarding the matter, and felt that it certainly was vastly better to remove the cartilage submucously. Moreover, he was convinced that it was a very troublesome and disagreeable thing to have even a bowing of the cartilage, and that the proper thing was to take out every cartilage that was not straight and in the median line. So he began to do this in many cases, and with beautiful results. In time he became convinced that the operation was unnecessary, excepting in certain rather exceptional cases. He still does the operation and likes it very much in these selected cases, but he does it in perhaps only ten per cent of the cases that he did it previously. Not that he had any disagreeable results or experiences with it, but simply because he could not morally feel that he was doing the right thing in subjecting the patient to this operation, excepting in cases of marked deflection, causing or nearly causing atresia.

What better operation is there in the simpler and more common cases, where there is a bowing of cartilaginous septum and some restriction of breathing on one side, or asthmatic symptoms? He uses a small, sharp saw—a Holmes or Pynchon saw—and removes the bony ridge and spur, regardless of the sacrifice of mucous membrane. In conjunction with this, he always does what he calls a "posterior inferior turbinectomy." He leaves the anterior part of the turbinate body alone, because he feels that under climatic influences it has a great deal to do with the physiology of the nose in a protective sense. But he saws off the entire posterior part of the inferior turbinate, to which is always attached in these cases a mulberry-like hypertrophy, the socalled "posterior hypertrophy." At the same time he snares off through the nose the mass of adenoids that almost invariably exists, not so much in the vault of the pharynx as in a vertical mass down the sides of the pharyngeal wall, like an exaggerated fold. This adenoid fold exists there because the serration has been faulty, and to remove it he passes the wire snare back through the nose until the end of the canula of the snare impinges on the basilar process of the occipital bone, the wire loop descending on the posterior wall of the pharynx, and then, engaging the adenoid mass in the loop of the snare by drawing the wire slowly home, it is snared off. This operation gives the patient a good nose—better, in his judgment, than the best submucous operation in these cases. He has not seen this method described by any rhinologist, and considers it a valuable one.

Regarding nasal packing after a submucous resection of the septal cartilage, he has tried the sponge packing, as described by Dr. Ingals, but not with the same fine technic, which he admires. But in his hands it was entirely inadequate. He found it so because he could not get an equal distribution of pressure over the operative field. He believes in packing, not because of the danger of hemorrhage, but for an entirely different purpose—a purpose which was not mentioned by the essayist, namely, in order to have perfect approximation of the bases of the perichondrium of the two sides. He always packs the narrow side of the nares only, that side in which the breathing space was restricted, and he uses ordinary loose mesh sterile gauze, previously cut in long strips and rolled like a little bandage. He always keeps a number of them on hand, all sterilized and kept in a glass jar ready for use. The gauze roll is moistened before using, which contributes to neat packing. Ordinary packing forceps are used. He packs only the side of septum convexity; he does not pack both sides, for fear of sloughing. The parts have already been wounded, and there is a certain amount of thrombosis of some of the septal veins, and if the nares were packed too tightly on both sides, necrosis might result. The packing is left in place only for twenty-four hours.

He quite agreed with the essayist in his ideas, which were progressive. The day of packing and leaving it in for as long as four days is past.

Regarding hemorrhage in submucous cartilage resection, he has never had much experience with it. There are no blood vessels cut, if the dissection has been properly done. Someone spoke of "delayed hemorrhage"—hemorrhage that occurs a week or ten days after operation. What does delayed hemorrhage mean? Delayed or secondary hemorrhage means wound sepsis, and nothing else.

Dr. A. M. Corwin said he was entirely open-minded with regard to the statement that an extensive septal operation, such as this is at times, can be done without packing, but on that point he was from Missouri. He had no reason to doubt the doctor's statements as to his results, but his own experience has been in accord with Dr. Freer's, that hemorrhage does occur occasionally, and for this packing is indicated. Furthermore, we operate on no other part of the body practically without dressing the wound, and we see no reason why we should make an extensive opening in the mucous membrane, cut away bone and cartilage, producing two loose flaps of membrane and perichondrium, and leave the wound unprotected against infection. The only case of abscess of the septum following resecting that he had ever seen after his own operating was in the case of a patient, entirely normal, with a perfectly clean nose, packed, and everything normal, who became nervous and dragged away the packing and left the wound open. An abscess of the septum developed at the site of the resection, and greatly increased her suffering and the danger and delay in healing. Moreover, there is no harm in packing, if done properly, while hemorrhage and infection may occur without it; therefore, the argument is in favor of packing.

He has found in the past few years that the use of a little gauze roller bandage and camphorated vaselin has been very satisfactory for this purpose. He cuts these strips of gauze, which are one-quarter inch wide, into two or three inch lengths, and boils them in the vaselin in a small crucible at the time of operation. This is cooled off by the time the operation is finished. These little pieces of gauze thus saturated are then packed carefully into the nose, one of them having been spread over the septal wound. These are left in practically until the second day. Then two or three pieces are removed. Each day following a few pieces are taken out at a time. There is no pain at all from removing them. This seems to him a very good way of packing, as he can take out what he wants and leave what he wants, without dragging upon long, unmanageable strips. The speaker believes that any man who does a radical operation on an inferior turbinate and does not pack, is almost certain to have hemorrhage, often profuse, sometimes serious, and he understood the writer to say that he employs little or no packing in such cases.

There were many points he would like to go into, but believed that we should accentuate the proposition that we should treat a nasal wound as far as possible in an aseptic manner. This involves proper protection by packing. We know that the larger the opening made, whether by incision or incidental tearing, the more need there is of packing.

To limit the discussion tonight to this one point of to pack or not to pack is unfortunate, since the subject matter of the paper has comparatively little to do with this point, notwithstanding the title, but is a lengthy dissertation on the technic of operating, and many interesting points touched upon by the essayist should be discussed, favorably or otherwise.

Dr. Edwin Pynchon said that while the essayist had very modestly disclaimed any credit for bringing anything new before the members, he must acknowledge that he had brought forth some ideas that were worthy of mention that had not been elsewhere brought out, and he wished to particularly speak of the use of the blunt pointed tenotomy shears.

Regarding packing, he has found that the method advocated by Dr. Freer is the most practical. However, in place of bismuth he likes to medicate it with a vaselin preparation containing antiseptics. One of the features to consider in packing the nose is that at the time of operation, owing to the use of cocain, the outer wall is very much contracted, and of course afterwards becomes congested, so for that reason it may make too much pressure. One of the great objects is mechanically to hold the two opposing surfaces together, and the more perfectly they are put in apposition, the more surely they will heal and thus avoid the formation of hematoma which can otherwise organize and cause ultimate thickening of the septum, while it was designed through the operation to cause the septum to be as thin as possible. So, consequently, he is in favor of packing both nostrils.

Regarding the removal of the packing, as Dr. Freer suggests, he is in the habit of taking out half of the upper part in both nostrils the day after the operation; then, maybe on the second day, all of the packing on the side that was concave, and on the third day the remainder of the packing. Prior to using these strips he used Bernays-Simpson tampons, but it is easy to see, from a mechanical standpoint, how impossible it is to make accurate apposition of the perichondria by the use of a device of that kind, which swells unequally (two from the same box will swell differently), and may thus produce too much pressure. Another thing: The tampon, introduced in any way, cannot cover as much area as has been separated, so consequently above or back of the tampon hematoma may occur, and any thickening of the septum is detrimental in the way of interfering with the breadthway in the upper part of the nose.

Dr. George E. Shambaugh stated that he had learned in the early days of his practice that it was not a safe procedure to do even the simpler operations in the nose and allow the patient to go home without packing, especially in a city the size of Chicago, where it often requires several hours before a patient can be reached. Both on account of the discomfort to the patient in having the nose packed, and because of the long trip from the office to the patient's home, Dr. Shambaugh has in recent years performed almost all of even the simpler operations of the nose at the hospital, where the patient is allowed to remain over night. He is not in the habit of packing the nose under these circumstances, for if a patient remains quiet in bed, the chances of a hemorrhage are much less than if he attempts to make a long trip to his home after the operation. If a hemorrhage should occur, the interne at the hospital can pack the nose, if necessary. As a matter of fact. Dr. Shambaugh can recall but one case where a patient left in this way had to have the nose packed by the interne; this was a case where nasal polypi and part of the ethmoid bone were removed.

In his operations on the septum he has always been in the habit of packing the nose. The packing in one side is removed at the end of twenty-four hours, and the other at the end of forty-eight hours. He places a piece of paraffin on the side of the septum where the mucous membrane has been incised, which facilitates very much the removal of the packing from this side.

The point which Dr. Vail wished to emphasize, that secondary hemorrhage always means sepsis, Dr. Shambaugh is not willing to accept. He stated that while it is true that sepsis is often the cause of secondary hemorrhages, it does not follow that all secondary hemorrhages are the result of sepsis. In most of the cases of secondary hemorrhages which he has seen develop in cases where the tonsils had been removed, the hemorrhage took place usually from five to ten days after the operation. In not a single one of these cases has he been able to recognize anything which he would class as sepsis, and in none of these cases was the local reaction at all marked. In a case of severe secondary hemorrhage from an operation in the nose, the bleeding was accounted for by a high blood pressure, and there was no evidence at all of infection in the nose.

Dr. Otis H. Maclay said he had enjoyed the paper and discussion, and especially the conservative remarks with referenec to septal work. It has always been his desire to so do his septal work that he could leave as much as possible of the septum, endeavoring only to get a functionating nose.

Regarding packing, he has always used packing, leaving it in originally for forty-eight hours. He has never had any sepsis, so far as he knows. For the last six months he has been taking out the packing, as a rule, in twenty-four hours. He has never had any hemorrhage that he has known of. There has always been some oozing and bleeding in all his operations. Whether without packing that would have been present or not, he does not know, but he has always felt safer when it has been used. He has packed less firmly in the last six months than before that time, but the packing, although it has allowed perhaps a little more oozing, has always been sufficient to control any bleeding that has occurred, but he has never felt perfectly justified in leaving it all out.

It is a matter of progress to hear this paper. Perhaps it is the proper course, and we may all gradually work into it. At least, the method should be carefully considered before being accepted or condemned. The papers read before the society and the discussions represent the best efforts in this line, and although the society should be progressive, it is equally important that we should be conservative in the methods employed, since the work recognized in the society is considered the proper and safe course to pursue, and doubtless is adopted by others on that basis.

Dr. J. Z. Bergeron thinks that the principal reason for not packing is largely, if not absolutely, to lessen the patient's discomfort caused by the packing. For the last five or six months he has used the strip packing described by Dr. Freer, one layer on top of another, surrounded by a layer of gutta percha, and on removing it, after twenty-four or thirty-six hours, he has found that it caused absolutely no pain.,

Dr. Edwin Pynchon said it had always occurred to him that in the use of the packing Dr. Freer advises, getting it in a piece and cutting off a strip about one-quarter or three-eighths inch in width, it is naturally irregular in shape, and if it is not cut in the right direction it breaks easily. It has seemed to him that some manufacturer should put this material on the market in strips with a selvage edge. He has never been able to find it.

Dr. Joseph C. Beck has, within the last three months, done quite a few submucous resections without packing. He would not say that he had left the nose without anything, but he had used no material such as gauze. He considers that the stoppage of the primary hemorrhage is very essential, and while he has the flaps open he packs the gauze firmly between them and allows it to remain in there from three to five minutes. Then, after removing it, he watches for a minute or two, until he believes that the hemorrhage has stopped. Of course, if the arteries that come from the palate to the nose are cut, they might possibly start another hemorrhage, but five minutes' waiting is sufficient time. When he is sure that the hemorrhage has stopped, he brings the parts together as firmly as possible, holds them and introduces the wedge-shaped metal spring, as recommended by Brüning, he believed.

Dr. Beck agrees with Dr. Shambaugh. He has not packed a nose after an inferior turbinate operation since he can remember, and has had no occasion to regret it.

He used the spring referred to above in ten successive cases, with good results. Then he had a case where a hematoma followed, the pain radiating to the face, with slight temperature. But he would not stop using the spring on account of this one case. Previous to using the spring, he used the packing referred to by Dr. Bergeron, which he (Dr. Beck) devised for that purpose, and had no occasion to regret it, except the discomfort occasioned to the patient.

Dr. Robert Sonnenschein answered Dr. Pynchon's question regarding his inability to obtain gauze with a selvage on both sides. It is used very extensively in Europe. Mr. V. Mueller carries it, and it can be obtained in the sizes of three-eighths and one-half inch.

Dr. Louis Ostrom, in closing, said that the members were behind the times if they packed. He has not packed for five years. They would not discontinue packing in Europe almost exclusively after submucous resection unless it was a good procedure. In the later books—St. Clair Thompson's and Parker's—published this year—no packing is advocated. Killian does not pack. Packing is not essential. He cited a case in which temperature had followed the operation, and after three days of this condition, with pain radiating to the face, he inserted a knife, expecting to see pus, but instead found only clear serum. He had a case of a hemophiliac in whom the blood dripped out like water, in which he packed. He gave five packages of antitoxin, and within an hour afterward had clotting. The packing was removed the next morning, and in about four hours afterward the nose was swelled just as tight as if there had been a packing there. He had tried out the methods of packing referred to over and over again, and has had nothing but trouble from them. When he has bleeding he packs, but is not going to pack until there is bleeding. He keeps his patients under control so that they are right beside him.

Regarding perforations, we must remember that our work today must not be classed with that done three or four years ago. Dr. Freer said he had had perforations by accident. Dr. Ostrom has had them, but they were due to carelessness, or things that were beyond his control. But in those conditions it was simply a puncture, just as though made for drainage purposes.

He has never heard of the wire springs, referred to by Dr. Beck, but is going to get some.

Regarding his use of the expression, anterior tip, it was simply a doubling up of language. He meant simply the tip or anterior end of the septum.

All the points brought out by Dr. Freer are covered in the paper, which the essayist did not have time to read in full. The paper deals only with the technic, not with the pathology, but every form of deflection is considered, and cases cited to explain the technic.

ANNALS

OF

OTOLOGY, RHINOLOGY

AND

LARYNGOLOGY.

VOL. XXII. SEPTEMBER, 1913. No. 3.

XXXV.

DISEASES OF THE LABYRINTH WITH A REPORT OF NINETEEN CASES.*

BY FRANK R. SPENCER, A. B., M. D.,

BOULDER, COLO.

A brief review of the anatomy and physiology of the semicircular canals may not be out of place before considering diseases of the labyrinth, especially since it has been only a few years that this branch of otology has been well understood. The horizontal or external semicircular canal lies almost horizontally, as its name implies. The ampulla is anterior, while the smooth end is posterior and a little lower, so that the canal is slightly oblique. The superior or frontal semicircular canal is almost vertical with its ampulla anterior and external, adjoining the ampulla of the external canal. The smooth end is, therefore, internal and posterior to the ampulla and unites with the smooth end of the posterior canal to form a common opening into the vestibule. The posterior or sagittal canal is also oblique, with its ampulla opening into the lowest and most posterior portion of the vestibule. I have given the positions of the canals, because it better enables one to understand the direction of the movement of the endolymph during the subsequent tests.

---

*Presented as candidate's thesis at the nineteenth annual meeting of the American Laryngological, Rhinological and Otological Society, held at Washington, D. C., May 8-10, 1913.

According to Ewald's law, the movement of the endolymph from the smooth end toward the ampulla is the more effective movement in the external or horizontal semicircular canal, and produces nystagmus to the same side. Conversely stated, the movement of the endolymph from the ampulla toward the smooth end is the less effective movement and produces nystagmus to the opposite side. If we consider the superior or frontal semicircular canal and the posterior or sagittal, the law must be reversed.

With Ewald's law in mind, we have at least the five following methods of testing the reaction of the labyrinth: First, the turning test. Second, the caloric test. Third, the finger test. Fourth, the fistula test, and fifth, the galvanic test. The turning test has the advantage of testing both ears at the same time, while its disadvantage is that each ear cannot be tested separately. However, if one labyrinth is more irritable than the other, the more irritable one determines the direction of the nystagmus. In case one labyrinth has been destroyed by disease or by an operation, we are able, of course, to test only the other internal ear by turning. By the caloric and galvanic we may test either ear separately or the two together.

Bárány has demonstrated that turning either to the right or to the left ten times produces the best reaction.[1] If we turn a patient with normal ears to the right ten times and stop, he should have, immediately after the turning, a horizontal nystagmus to the left for fifteen to thirty seconds—that is, the quick component should be directed to the left. Conversely, after turning to the left ten times and stopping, there should be a horizontal nystagmus to the right for fifteen to thirty seconds. To test the external or horizontal semicircular canals by turning, the patient should sit upright in a straight back revolving chair with the head erect. The superior canal may be tested in a similar manner, if the patient inclines his head forward during the turning, and the posterior if he inclines his head backward, so as to have the head in either position in the horizontal plane. The last two canals produce, of course, a rotatory nystagmus. In Prof. Urbantschitsch's clinic in Vienna (which was formerly in charge of Politzer) Bárány, Neumann, Ruttin and others use a light weight revolving arm chair with an iron rod extending from the back of the seat up through the back of the chair for a distance of one meter above the top of the back. This rod is about two and one-half centimeters in diameter and is grasped by the physician above the patient's head during the turning. It is quite important to have each of the ten turns of uniform rapidity and to stop the patient as quickly as possible at the end of the tenth turn. The patient should wear dark smoked glasses during the turning.

The caloric test may be made with either hot or cold water, if the drums are intact or if suppuration is present. If there is a perforation with an absence of pus (healed chronic purulent otitis media), hot and cold compressed air may be used in order to avoid moisture. Syringing with cold water normally gives nystagmus to the opposite side, and with hot water to the same side. Cold water is usually more agreeable to the patient and should have a temperature of about twenty-five degrees centigrade, although it may be necessary to use water of fifteen degrees in some cases in order to obtain the reaction. The hot water should have a temperature of about forty-eight degrees centigrade.

The finger test may be made after syringing the ear. After cold syringing the patient falls toward the syringed ear, if asked to stand erect with the eyes closed, and misses the finger of the physician by passing his finger to the left of the observer's if the left ear was the one syringed. The above test is the reaction of a normal ear to cold syringing, and the condition would be reversed if hot water were used.

The test for a fistula may be made by either compression or aspiration. A fistula is usually located in the bony portion of the horizontal or external semicircular canal, as this is the most exposed of all the semicircular canals to middle ear suppurations. However, it may be in the cochlea, vestibule or one of the other semicircular canals. Ruttin states that in forty out of fifty cases in which a fistula was demonstrated, compression of air in the external auditory meatus and middle ear produced nystagmus to the same side, and aspiration produced nystagmus to the opposite side. In other words, this is the typical reaction of the labyrinth to condensation and rarification of air in the middle ear, and is the rule. This shows that the endolymph is driven by compression from the smooth end toward the ampulla, which is the more effective movement for the horizontal semicircular canal and, therefore, produces nystagmus to the same side. If we use the fistula test while the patient is under the influence of a general anesthetic on the operating table, we produce only the slow component from the labyrinth, as the quick or central component is absent during the third stage of ether or chloroform anesthesia. The author wishes to emphasize the importance of making the test for the fistula symptom, first, because it yields, when present, valuable information, and, second, it is an easy test to make. If present in any case of chronic purulent otitis media, we are almost certain to find a cholesteatoma in the middle ear and antrum as the etiologic factor in producing the erosion of the outer bony labyrinth.

The four tests just given all act on the endolymph in the labyrinth, while the galvanic test determines the reaction of the eighth nerve. If we place the cathode in the right meatus we should expect a nystagmus to the right, and if we place the anode in the right meatus we should expect a nystagmus to the left, if the auditory nerve is normal. The test does not depend upon an intact labyrinth, as it may be used soon after the labyrinth has been destroyed either by disease or by an operation. We may begin the test with four milliamperes of the galvanic current, but as much as eight to fifteen may be required before the nerve responds. If the nerve has been destroyed or is atrophic, it will, of course, not respond.

Ruttin has devised several pieces of apparatus for the above tests. The first consists of two aural specula mounted on opposite ends of a semicircular rubber headband so that the headband holds the two specula firmly in the ears. A small metal tube is fixed in each speculum so that it can be connected with a rubber tube and a jet of either hot or cold water thrown into the external auditory meatus from either one or both sides. The return flow passes out through the specula. This piece of apparatus makes it possible to syringe each ear separately with a uniform quantity of water equally divided between the two ears. Of course, if two normal ears in the same patient are syringed simultaneously with either hot or cold water, there will not be any nystagmus produced, as the reaction of one labyrinth counteracts the reaction of the other. A similar piece of apparatus has been made so that a metal electrode is held firmly in each external auditory meatus by the rubber headband, so that the reaction of either the right or left auditory nerve may be tested without changing the apparatus.

Three degrees of spontaneous nystagmus are recognized: First, spontaneous nystagmus when the patient looks in the direction of the quick component, but which is absent when the patient looks either straight ahead or in the direction of the slow component. Second, spontaneous nystagmus is present when the patient looks straight ahead, but is increased by looking in the direction of the quick component. The nystagmus disappears, of course, when the patient looks in the direction of the slow component. Third, the third degree is present in all positions of the eyes, but is greatest when the patient looks in the direction of the quick component, and is least when the eyes are turned in the direction of the slow component. Sudden destruction of one labyrinth always produces third degree nystagmus with the quick component directed away from the diseased ear. The slow component is the peripheral one, and is due to irritation of the labyrinth, while the quick component is always central and represents nature's effort to counteract the naystagmus due to labyrinthine irritation. For instance, if we syringe the ear of an unconscious patient we produce only the slow component, as the central or quick movement of the eyes is absent. This can be well demonstrated during the third stage of chloroform or ether anesthesia by syringing the ear just prior to a mastoid operation. The eyes move in the direction of the slow component and remain in that position as long as the syringing is continued, because without the central component we cannot produce nystagmus. Inflammations and suppurations of one side produce a nystagmus to the opposite side for from three to fourteen days.

Ruttin (Ruttin's Course in Surgical Diagnosis, University of Vienna. Similar table given in Prof. Urbantschitsch's Lehrbuch der Ohrenheilkunde, fifth edition, page 456) gives the following table or classification of the different forms of labyrinthitis: First, cases of circumscribed serous labyrinthitis give a history of labyrinthine symptoms during the attacks. There is usually horizontal and rotatory nystagmus to the opposite side, but rarely nystagmus may be directed to the same side. Hearing is present; the fistula symptom is present; and the labyrinth reacts to the caloric test. Second, cases of diffuse serous labyrinthitis do not give any history of labyrinthine symptoms during the course of the middle ear disease. Third degree nystagmus is present with the quick component directed toward the opposite side usually, although it may be directed toward the same side in mild cases. Hearing is usually absent, but may be present. The ear usually reacts to the caloric test, but there may be a total absence of any reaction. The fistula symptom is usually absent, but is present in those cases in which the diffuse serous labyrinthitis was preceded by a circumscribed serous labyrinthitis. Third, cases of acute purulent diffuse labyrinthitis do not give any history of labyrinthine symptoms during the attack of middle ear disease. Third degree nystagmus is present with the quick component directed toward the opposite side. The hearing, the caloric reaction and the fistula symptom are all absent. Fourth, cases of chronic diffuse purulent labyrinthitis give a history of labyrinthine symptoms during the attack. Spontaneous nystagmus is often absent, but may be present. The hearing, the caloric reaction and the fistula symptom are all absent. The first two types of labyrinthitis demand only a radical mastoid operation, while the third and fourth types demand a radical operation and an operation upon the labyrinth too.

Case 1.—Mr. A., age twenty-five years, examined June 29, 1911. On the twenty-seventh day of the preceding month he had had a radical mastoid operation on the right side and made an uninterrupted recovery. For a few days prior to the examination he had had severe vertigo, and the fistula symptom was found to be present upon examination. Compression produced nystagmus to the same side, and aspiration to the opposite side. Examination of the eyes showed a spontaneous nystagmus with the quick component directed to the right when the eyes were turned to the right. The nystagmus was made stronger by moving the head, which shows that he was not a malingerer. He showed a slight nystagmus to the right after syringing, but did not fall in the direction of the slow component to the left, as we would expect. This latter test was made ten minutes after the syringing, so it is not as valuable as if made immediately after the syringing. Examination of the patient's hearing showed the following: The patient heard a conversational voice at one-half meter with the right ear; Weber's and Rinné's tests were negative; and bone conduction was slightly shortened. The upper tone limit was below normal, and the lower tone limit was elevated. Turning the patient to the left ten times produced nystagmus to the right which lasted thirty seconds. Turning to the right ten times produced nystagmus to the left for twenty-seven seconds. Syringing the right ear with cold water produced nystagmus to the left.

Diagnosis.—Diffuse serous labyrinthitis following circumscribed serous labyrinthitis. As he had had a radical mastoid operation, he did not need any operation, but he should be watched to see if a purulent labyrinthitis develops. There are three types of serous labyrinthitis so far as the hearing is concerned: First, cases with good hearing. Second, cases with poor hearing, and third, intermediate types with only a moderate preservation of hearing.

Case 2.—Mr. B., age eighteen years. Right ear normal. The history of the left ear is as follows: Eleven years ago, in the province of Galicia, he had a Stacke radical mastoid operation on the left side. After the operation the discharge ceased. One year ago the left ear began to discharge again, but he had not had any headache, vertigo or vomiting. Examination of the left ear showed the following: He heard a conversational voice at one and one-half meters; Weber's and Rinné's tests were negative. The lower limit was elevated and the upper tone limit was below normal. By Schwabach's test the bone conduction was not shortened. The caloric reaction was present; polypi and granulations were present in the antrum. Turning to the right ten times produced nystagmus to the left for fifteen seconds; turning to the left ten times produced nystagmus to the right for thirty seconds. Upon testing for the fistula symptom the patient showed nystagmus to the same side with compression, and to the opposite side upon aspiration.

Diagnosis.—Circumscribed serous labyrinthitis of the left ear. A radical mastoid operation is indicated. The prognosis should be guarded, as there is some danger of a purulent labyrinthitis following the radical exenteration.

Case 3.—Mr. C., age thirty-four years, gave a history of chronic purulent otitis media of the right ear for the past seven years which followed influenza. One year ago the left ear began to discharge. The patient complained of headache, vertigo, nausea and vomiting recently. Examination of the left membrana tympani revealed a dry perforation in the posterior superior quadrant leading into the antrum. Examination of the hearing showed the following: A conversational voice was heard at one-fourth meter with the right ear and at two meters with the left ear; by Weber's test the fork was heard better in the left ear; Rinné's test was negative for both ears. The lower limit was slightly above normal, but the upper limit was normal. Spontaneous nystagmus was absent. The fistula symptom was present in the left ear with nystagmus to the left by compression and to the right by aspiration. Turning to the right ten times produced nystagmus to the left for thirty seconds. Turning to the left ten times produced nystagmus to the right for thirty-one seconds, which was normal for both labyrinths. As the vertigo in this case came on only fourteen days ago, and since his hearing is as good as before, he has only a fistula without any disease of the membranous labyrinth.

Diagnosis.—Circumscribed serous labyrinthitis. In this case only a radical is indicated, although we may treat the patient for awhile, as the fistula may close without an operation. If the patient is not treated he is almost sure to have a purulent labyrinthitis.

Case 4.—A young lady gave the following history: Two months ago she had a purulent discharge from the left ear, and a few days ago she began having a discharge from the right ear. She thinks she has tuberculosis, as she has been given injections and, as she does not know what the injections are for, she has been led to believe she is getting tuberculin. From her statements it was evident that she had been taking mercury, and "606" may have been injected in place of tuberculin. She stated that her hearing failed rapidly after the injections, so this would lead one to believe she had been given "606." The hearing tests were as follows: The air conduction was shortened for both ears, but the bone conduction was excellent. She heard reasonably well with each ear when the two and one-half meter speaking tube was used. Weber's test was lateralized in the left ear and Rinné's was positive for each ear, but more positive on the right side. The upper tone limit was below normal for each ear and the lower tone limit was elevated on each side. Third degree spontaneous nystagmus was present, which was most marked when the patient's eyes were directed to the left. Turning to the left ten times produced nystagmus to the right for fifty seconds; turning to the right ten times produced nystagmus to the left for thirty-five seconds. Cold air passed into both the right and left ear successively gave negative results. With the right ear the patient heard forks D 1 to C 4, and with the left ear forks A 1 to G 3 of the Bezold-Edelman series, when the hearing should have been perfect for forks C 2 to C 8. The patient has, therefore, only the middle range of hearing. Judging from the history, the internal ear disease must have preceded the middle ear disease. The vestibular division of the eighth nerve is negative, as it reacts well, but the cochlear division is diseased.

Diagnosis.—Chronic purulent otitis media and toxic neuritis of the cochlear division of the auditory nerve in each ear. The prognosis is better if it is due to syphilis, but in this case it may be tuberculous. Lead, quinin and the salicylates have a selective action on the cochlear nerve. The patient formerly had large doses (0.5 g.) of aspirin for a prolonged period, so that the toxic neuritis may have been due to aspirin.

Case 5.—A boy, age seven years, was admitted to the otologic department of the Vienna general hospital May 15th, with the following history: Three weeks ago he had pain in the left ear, and three days ago very severe pain in the left ear and headache, but without nausea, vomiting and vertigo. Two days before admission he had a high temperature. The left membrana tympani was red and bulging and the landmarks were obliterated. The posterior superior wall of the external auditory meatus was bulging and the mastoid process was tender upon deep pressure. The right ear was negative. Functional examination of the left ear revealed the following: He heard a conversational voice from four to five meters and a whispered voice at a distance of two meters. Weber's and Rinné's tests were negative; bone conduction was lengthened. He could not hear fork C 1, but did hear fork C 4. The turning tests were normal.

Diagnosis.—Acute purulent otitis media. A free incision was made in the left membrana tympani on the day of his admission to the hospital. The temperature before admission was 39.2° C., and after the incision it fell to normal by lysis within the next few days. We may have fever if the pus extends to the dura or lateral sinus, but we are not apt to have fever from an uncomplicated middle ear abscess with good drainage. The ninth day after his admission the temperature was 38.5° C. At this time (ninth day) a simple mastoid operation was performed. The pus came from the posterior portion of the mastoid under high pressure, so that the patient had either a perisinus or extradural abscess. The lateral sinus was exposed over a large area, and a small incision was made in the sinus wall, but no thrombus was found. After the operation the fever continued with a very slight fall until four days later, when it rose to 39.6° C. A second operation was decided upon May 30th. The internal jugular vein was ligated on the left side and a thrombus removed from the left lateral sinus. The temperature gradually dropped, reaching normal on June 4th. At this time there was free drainage through the sinus, as fluid injected into the upper portion of the internal jugular above the point of ligation passed out through the opening in the lateral sinus. On the eighth day the temperature was high again, and it was impossible to find the lower end of the upper portion of the jugular, so a bulb operation was performed. (The spinal accessory nerve is over the jugular vein in sixty per cent of all cases, and it was over the jugular in this case. The bulb was laid over the nerve from above, so that the spinal accessory is now under the jugular.) Since this last operation the recovery has been perfect. The infection in this case was due to the streptococcus pyogenes, and this organism is apt to produce a sinus thrombosis.

Case 6.—A girl, age seventeen years, was admitted to the otologic department of the Vienna general hospital the day before this examination with the following history: Eight days ago she had a severe sore throat, and two days later severe pain in the left ear, with a temperature of 37.5° C. to 38.8° C. The left membrana tympani had a small perforation in the anterior inferior quadrant. The mastoid was tender upon deep pressure, especially over the tip, and the neck was rigid. Ruttin stated that the torticollis might be due to, first, Bezold's type of mastoiditis; second, enlarged lymph glands; third, peribulbar abscess; fourth, thrombosis of the mastoid emissary vein, and, fifth, Gleissenger's sign. If pus is present around the bulb, it is likely to pass into the deeper structures of the neck and produce torticollis: If such is the case the pus must be evacuated by going down to, but not into, the bulb. The functional tests for the left ear showed the following: Conversational voice heard at thirty centimeters; a whispered voice at five centimeters. By Weber's test the sound was lateralized in the left ear and Rinné's test was negative. The air conduction for forks C 1 and C 4 was shortened.

Ruttin stated that this patient might have a brain abscess which, if present, must be a temporal lobe abscess. If the abscess were located in the cerebellum she would have vertigo and nystagmus with a disease of the labyrinth, which she did not have. She did not have aphasia, so she probably did not have a temporal lobe abscess. In the latent stage, without any focal symptoms, there is a slow pulse; no evidence of meningitis, as the reflexes are normal, and no dermagraphia. This patient must be kept under observation for a time before a positive diagnosis can be made.

Case 7.—A young lady of about twenty years of age, was admitted July 13th with the statement that she had been totally deaf in the right ear since early childhood and that recently she had lost a great deal of hearing in the left ear.

Upon testing her hearing the right ear was found to be totally deaf and the left showed the following: The conversational voice was heard from one-fourth to one-half meter. Weber's test was lateralized to the left; Rinné's was negative, and the bone conduction was shortened. She heard forks C 1 and C 4.

The eyes showed a spontaneous nystagmus of the third degree, which was probably ocular, as she had astigmatism in each eye with convergent strabismus and amblyopia of the right. Turning to the right produced nystagmus to the left of questionable duration, as the squint and ocular nystagmus greatly interfered with the test. Turning to the left ten times produced nystagmus to the right for twenty-four seconds. The finger test was negative. The right ear did not react to the caloric test, but the left reacted normally. The galvanic test was made, beginning with four milliamperes and increasing to fifteen. However, the ocular nystagmus interfered so much that it was impossible to tell very much if anything about the accuracy of the reaction.

Diagnosis.—Congenital defect of the eyes (retinitis pigmentosa with a history of parental intermarriage) and of the vestibular and cochlear apparatus.

Case 8.—A man, fifty-four years of age, was admitted July 14th with poor hearing. In the eye clinic a diagnosis of papillitis due to suspected brain tumor was made. He had been ill for five weeks and complained of tinnitus in the left ear, but he was not very intelligent, so he could not tell what symptoms he had.

The hearing tests showed the following: He heard a conversational voice at a distance of seven meters with the right ear and at a distance of five with the left; the whispered voice at five meters with the right and at two with the left. Weber's test was not lateralized (Weber's in the head) and Rinné's was positive for each ear. The bone conduction was normal for each ear, and he heard forks C 1 and C 4 with each ear. The cochlear apparatus may be slightly affected on the left side.

Spontaneous nystagmus was absent, but the following reactions were obtained from the labyrinth: Turning to the left ten times produced nystagmus to the right for twenty-four seconds, and turning to the right ten times produced nystagmus to the left for twenty-five seconds, so both tests were normal. With the finger test, after turning to the right ten times, the patient's finger deviated to the right, and after turning to the left ten times, the patient's finger deviated to the left. This test also produced a normal reaction. For the caloric test both ears were first syringed simultaneously with cold water. For this test both ear drums must, of course, be present. There was an absence of any reaction and the test was negative. Syringing the right and left ears separately with cold water gave a normal reaction, except that the left ear was a little more irritable than the right. However, the reaction was typical, normal or physiologic.

A brain tumor in the middle fossa is not apt to affect the hearing unless the tumor is large. If it is in the posterior fossa it is very apt to affect the hearing and the vestibular apparatus very early. Tumors of the cerebellopontine angle give rise to symptoms early from the cochlear and vestibular apparatus. All tumors of the brain produce shortened bone conduction just as soon as they are large enough to give rise to increased pressure. The membrana tympani may be slightly retracted, thus indicating increased bone conduction, but we find shortened bone conduction if there is increased intracranial pressure.

As this man has not spontaneous nystagmus he cannot have a brain tumor of any size, especially in the posterior fossa. If we find nystagmus due to a severe labyrinthine disease, the nystagmus disappears in from three to fourteen days. If the nystagmus is due to a cerebellar tumor, it occurs in attacks, and it may occur to either side with great irregularity. For instance, the direction of the quick component may change from the right to the left, or vice versa, while we are making the examination. However, the nystagmus is always present if the tumor is of any size. This patient cannot have a cerebellar tumor, as the reaction of the labyrinth is normal, unless the tumor is too incipient in its development to produce symptoms at this time. We must have this case return in two months for reexamination before we can be absolutely certain of the diagnosis.

Case 9.—A man in the employ of one of the steam railroads fell from the top of a box car and struck on his head. He was unconscious for fully three minutes and had a hemorrhage from both ears, but he did not have either tinnitus or deafness. The blood may come from any one of the following: First, the external meatus; second, membrana tympani; third, the middle ear; and, fourth, the labyrinth. As he had neither vertigo nor deafness, he did not have a fracture of the internal ear. He did not have spontaneous nystagmus. However, the accident occurred five days before this examination on July 15th, so if the nystagmus lasted only three days after the injury, it would not have been present at the time of the examination. With many fractures at the base of the skull we find a facial paralysis, because with a fracture of the petrous portion of the temporal bone we have involvement of the vestibule, cochlea and aqueductus fallopii. If only the tip of the petrous portion of the temporal is involved, the above three escape injury. He was able to hear a conversational voice at a distance of one and one-half meters with the left ear only. If he had only a slight fracture of the cochlea, the endolymph may have regenerated in five days with return of a moderate degree of hearing as a result.

Turning to the right ten times produced nystagmus to the left for thirty seconds, and turning to the left ten times produced nystagmus to the right for twenty-four seconds. This is a normal reaction of the labyrinth, so it is hardly necessary to test the internal ear with the tuning forks.

Concussion of the labyrinth cannot be demonstrated, so we should not make such a diagnosis; but fracture can be demonstrated both before and after death. (Please do not make a diagnosis of concussion of the ear or of the base of the skull. Ruttin.)

As this patient had a bandage under his chin and had lost several teeth as a result of the injury, we were forced to conclude that he fell on his chin and not on the top of his head. The force of the blow was transmitted from the inferior maxilla to the anterior wall of the external auditory meatus, resulting in a fracture of the latter. This accounts for the blood in the meatus. However, pus was present with the blood. He may have had either an acute or chronic purulent otitis media before the accident. (Ruttin thinks such cases should be operated upon, and he related a case to illustrate his point.) However, in the present case it is better to wait and pack the external meatus with gauze after dry cleansing, as the fracture, in this case, in all probability, did not involve the middle or internal ear.

Diagnosis.—Fracture of the anterior wall of the external auditory meatus. (The X-ray was to have been used, but the author did not learn what it revealed.)

Case 10.—A boy of six. He had acute purulent otitis media about eight months ago. Three weeks after the onset of the acute attack he developed sepsis, spontaneous nystagmus and vertigo with loss of vestibular reaction and hearing. His temperature at this time was 39° C., so that one was forced to think of either a purulent labyrinthitis or a sinus thrombosis. If he had a purulent labyrinthitis he would have a meningitis, and there were no meningeal signs or symptoms. On the other hand, we were not apt to find a purulent labyrinthitis in this case, as the otitis is acute.

Diagnosis.—Acute serous labyrinthitis.

Case 11.—Male, age thirty-four years. A history of chronic purulent otitis media of the right ear for the past seven years, and of the left for the past year. Recently he has been having headache, tinnitus, nausea, vomiting, vertigo, etc.

Examination of the left ear revealed a dry (?) perforation which led into the antrum just posterior to the short process. He heard a conversational voice at a distance of one-fourth meter with the right ear and at two meters with the left. By Weber's test the sound was lateralized in the left ear. Rinné's test was negative for both ears, and bone conduction was lengthened for both. The hearing for fork C 1 was shortened, and for C 4 it was normal. Spontaneous nystagmus was absent.

In testing for the fistula symptom in the left ear, nystagmus was produced by compression, with the quick component to the left, and nystagmus to the right by aspiration, which is the typical reaction and is with the rule. The fistula test failed to produce a reaction on the right side. If we obtain only the slow component of the nystagmus by the test, it should be directed to the opposite side in the typical and to the same side in the atypical cases. In the absence of the central control (cerebral control), as under ether or chloroform anesthesia, we obtain only the slow or peripheral (vestibular) component. The fistula may be single or multiple. The horizontal canal is involved in ninety per cent of the cases in which suppuration is present in the antrum. In other cases the fistula may be present in the frontal canal, oval window, round window or cochlea.

Turning to the right ten times produced nystagmus to the left for thirty seconds, and turning to the left ten times produced nystagmus to the right for thirty-one seconds. This, of course, was a normal reaction.

As the vertigo manifested itself in this patient only fourteen days prior to this examination, and as his hearing was as good as it had been previous to this, he has only a fistula in the bony labyrinth, without a disease of the membranous labyrinth.

Diagnosis.—Circumscribed serous labyrinthitis. Therefore, in this particular patient only a radical mastoid operation is indicated, and not an operation on the labyrinth, too. However, as his hearing is poor in the other ear, we may treat him for awhile. Without treatment he is almost certain to develop a purulent labyrinthitis. We must attempt to close the fistula with either fibrous tissue or bone.

Case 12.—A boy of six years presented himself for examination. He gave a history of acute purulent otitis media in the left ear and of chronic purulent otitis media in the right ear. His head was inclined slightly to the right, and the right side of his neck was stiff. The hearing in the left ear was almost perfect, but was very poor in the right. The patient had been vomiting that day and was very dizzy. His temperature was 39° C. Further testing revealed destruction of the right labyrinth.

At this time we have to think of the following causes of torticollis: First, meningitis; second, cerebellar abscess; third, temporosphenoidal abscess, if far back in this lobe of the brain; fourth, sinus thrombosis; fifth, perisinus abscess; sixth, Bezold's type of mastoiditis; seventh, meningismus; eighth, endocarditis.

This patient's nausea, vomiting and vertigo may have been due to a cerebellar abscess. The temperature of 39° C. may mean a sinus thrombosis. With meningitis, the higher the fever the greater the torticollis. The destruction of the labyrinth may also explain the nausea, vomiting and vertigo. A sinus thrombosis would account for the fever and torticollis. Ruttin thought a sinus thrombosis combined with a serous labyrinthitis the most probable cause. Therefore, he performed a simple mastoid operation with sinus exposure. An abundance of granulations covered the sinus, but the sinus was not opened. Two days later the sinus was incised because the temperature continued high. At this time the internal jugular vein was also exposed, as it should be, and two infected lymph glands were found lying over the internal jugular between the thyroid and facial veins. This part of the vein had, in all probability, been infected by the suppurating glands. A resection of the internal jugular vein between these two contributing veins proved this to be the case, as a thrombus was found here and not in the lateral sinus.

Diagnosis.—Otitische lymphadenogene Leptico-Pyanne, or infection of the internal jugular vein from infected glands. The lymph glands around the internal jugular are in the chain from the middle ear and external auditory meatus. This patient made an uninterrupted recovery, but he is deaf in the right ear, because the right labyrinth was destroyed by a severe type of serous labyrinthitis.

Case 13.—For the past five weeks the patient has suffered with attacks of vertigo, nausea, vomiting, diarrhea, formication, tinnitus and deafness. The last symptom was present only on the left side. The membrana tympani was negative on each side and spontaneous nystagmus was absent. The functional tests showed the following: The conversational voice was heard at a distance of five meters with the right ear and at two meters with the left; the whispered voice at four meters with the right and not at all with the left. By Weber's test the sound was lateralized in the right ear, and Rinné's was positive for each ear. Bone and air conduction were shortened for the left ear.

Upon using the finger test the patient's finger deviated to the right of the observer's before the turning test had been made to the right and after turning to the left, but the deviation to the right was greater after turning to the right. Syringing the left ear with cold water produced a slight nystagmus to the right.

If a brain lesion were present in this case it must have been on the left side, as the left ear was the diseased one and the lesion was in the cerebellum.

Diagnosis.—Increased irritability of the labyrinth due to a disease of the left posterior fossa or left cerebellar lobe, but we do not know the exact nature of the lesion from the data which the case affords.

Case 14.—A woman of twenty-two years was admitted with the following history: Two weeks before her admission to the hospital she developed a full feeling in the right ear, followed in three days by severe, deep, boring, constant pain. A free incision was made in the right membrana tympani the day the pain developed and in three days the pain had ceased. The day before her admission, or thirteen days after the onset of the disease, she developed severe pain in the right middle ear and mastoid process, immediately followed by nausea, vomiting and vertigo. Her temperature on the day of admission, at 10 a. m., was 37.6° C.

Examination of the right ear revealed a red membrana tympani with pulsating pus filling a perforation in the anterior inferior quadrant. The mastoid was tender, upon deep pressure, over the antrum and tip. The left ear was negative while the right was quite deaf. Rinné's test was negative for the right ear, but fork C 1 was not heard. The hearing for fork C 4 was shortened. The right labyrinth gave a normal reaction to the caloric test. In addition, the patient had diffuse bronchitis and was in the sixth month of pregnancy. There was also a periarticular abscess above the left hip joint.

During the subsequent twenty-four to forty-eight hours the mastoiditis underwent resolution, due to the application of cold, etc. However, the fever, bronchitis and periarticular abscess continued, so that it was necessary to think of a lateral sinus thrombosis with metastases in the lungs and hip joint, even though the mastoiditis had undergone resolution.

On the third day an exploratory operation was performed and the lateral sinus was exposed. Although the right ear was quite deaf, there was an absence of spontaneous nystagmus and the labyrinth responded normally to the caloric test. The deafness was, in all probability, toxic, so there was not any indication for a labyrinth operation. The lateral sinus was found far forward and was exposed above, but was perfectly healthy. Ruttin did not feel justified, under the existing circumstances, in ligating the internal jugular and exposing more of the sinus. For the same reason he did not open the antrum, as he found both the mastoid process and lateral sinus normal. He prefers to leave the wall of hard protecting bone, in such cases, between the antrum and mastoid tip.

Diagnosis.—Osteopyogenea or pus following the venous blood into the circulation.

Case 15.—This patient was admitted to the hospital February 18, 1905, with a history of headache, coryza, bronchitis and acute purulent otitis media of the right ear during the two weeks previous to admission. The mastoid was tender upon deep pressure and the right membrana tympani red and perforated. The functional tests showed only a middle ear disease, as the labyrinth was normal. The temperature was elevated. As the fever and other symptoms of mastoiditis continued for thirty-six hours, in spite of the abortive treatment, a simple mastoid operation was performed. The patient made a good recovery following the operation.

In February, 1911, the patient discovered pus discharging through a retroauricular opening. The discharge continued with more or less irregularity until he came back to the hospital in July, 1911. Two days before admission (July, 1911) he suffered from vertigo, nausea, vomiting and chills. The functional tests and the physical examination showed only an acute exacerbation of a chronic purulent otitis media.

In this case we have to think of an old circumscribed serous labyrinthitis since the operation in 1905, because upon closer questioning we find that he had had vertigo since two months after the operation. The granulations over the horizontal or external semicircular canal may have destroyed the outer wall of the bony labyrinth.

In July, 1911, a radical mastoid operation was performed by one of the youngest assistants in the ear clinic, but unfortunately the operation was followed by facial paralysis. A great many large cells were found in the mastoid, filled with pus, but the dura and lateral sinus were not exposed. For four days after this second operation the patient ran a septic temperature, so that on the fourth day a third operation was performed for exploration of the dura and lateral sinus. The dura was negative upon pressure, but the jugular bulb was covered with granulations. These were easily wiped away with a piece of sterile gauze, and the blood flowed freely from an accidental tear in the wall of the bulb inflicted by the gauze.

After the third operation the septic temperature continued, but was higher than before, without even returning to normal at any time during the twenty-four hours. This meant, of course, a complicated case of sepsis. For instance, erysipelas complicating sepsis of aural origin will produce such a temperature. A septic temperature due to a lateral sinus thrombosis only always drops below normal before each rise. We must think, too, in this connection of meningitis as a complication. However, further examination and investigation revealed an abscess in the region of the left tibia.

At the time of the third operation the right internal jugular should have been ligated, as after this the shock was too great to permit a fourth operation even under a local anesthetic.

Diagnosis.—Mural thrombosis in the lateral sinus and possibly a brain abscess.

Case 16.—This patient, a man of fifty years, complained of inability to hear well for the past twenty-five years, but he has always considered the left the better ear. For many years he has had tinnitus of both ears. Three or four months before his admission he developed vertigo, but this symptom ceased after taking potassium iodid. Fourteen days before this examination the left ear suddenly became worse. He admitted having had gonorrhea, but denied syphilitic disease.

The functional tests showed absolute deafness in the left ear and almost total deafness in the right. Spontaneous nystagmus was absent. Turning to the right ten times produced nystagmus to the left for thirty-two seconds, and turning to the left ten times produced nystagmus to the right for twenty-seven seconds. The finger test was negative. The right ear failed to react to the caloric test, and the left reacted a very little, if any.

Upon further inquiry the patient stated that he was in the habit of drinking five liters of beer a day and of smoking three cigars. Before he became totally deaf he was immediately made worse if he drank and smoked.

Diagnosis.—Toxic neuritis of the cochlear division of the auditory or eighth nerve. (Alcoholic neuritis.)

Case 17.—A boy of seventeen years complained of loss of hearing for the past two years with vertigo, but without tinnitus. His deafness was accompanied by epileptic seizures from the first, and his eyes showed a slight indefinite ocular nystagmus.

He heard only a loud conversational voice with each ear, but was unable to state with certainty in which ear he heard the fork better by Weber's test. Rinné's test was negative for each ear, and the bone conduction was shortened. It was doubtful whether he heard fork C 1 with either ear, and he heard fork C 4 only a very short time. He heard a conversational voice through the two and one-half meter speaking tube with each ear. Each membrana tympani was normal.

Since he had an indefinite ocular nystagmus, it was necessary to observe this carefully under smoked or colored glasses before making the turning test, so as to recognize any change in the nystgmus after the turning. Turning to the right ten times produced nystagmus to the left for thirty-nine seconds, and turning to the left ten times produced nystagmus to the right for forty seconds. The finger test was important, too, in this case, because he had a disease of the brain (epilepsy). The test was made, but was normal.

Diagnosis.—Otosclerosis of each ear. The increased irritability of the labyrinth was due to epilepsy, and the disease of the cochlea to the otosclerosis. The boy had congenital amblyopia (reported from the eye clinic), ocular nystagmus and epilepsy, so he is a degenerate.

However, the family history, so far as the ears are concerned, was negative.

Case 18.—A young lady of twenty-three years came to the clinic, at the request of her family physician, for an examination of her ears. She insisted that her hearing was perfect, but her physician felt an examination was necessary. She gave the following history: Two years ago she had an attack of optic neuritis in the left eye, which yielded to treatment with potassium iodid. Three weeks ago she had a recurrence of the inflammation in the same eye. She complained of a severe headache throughout the entire head, which she could not localize at any point. In addition, she had intestinal catarrh.

The physical examination of her ears revealed a normal ear drum on each side. The functional tests showed the following: The conversational voice was heard at a distance of ten meters and the whispered voice at five meters with each ear. Rinné's test was positive for each ear, but Weber's was indefinite. Bone conduction was normal, and she heard forks C 1 and C 4 perfectly. Spontaneous nystagmus was present and was most pronounced when she looked to the left. Her nystagmus was purely horizontal and was central, but a combined horizontal and rotary may be either central or vestibular or both. As it would be very hard to tell about a horizontal nystagmus produced by turning with the head erect, in this case the caloric test was used, as this test produces rotatory nystagmus. After syringing the right ear with cold water she had a rotatory nystagmus while looking straight ahead. However, the horizontal nystagmus did not disappear. With central nystagmus we cannot tell whether it is first, second or third degree. When central nystagmus is present the movement is quite pronounced upon looking either to the right or to the left, but it is absent while looking straight ahead. With central nystagmus we may have the third degree without the first or second, which is impossible with a vestibular nystagmus. In other words, with a third degree vestibular nystagmus we always have the first and second degrees. This patient vomited spontaneously and without nausea just before the syringing, but not during or after the syringing, so we are dealing with a central lesion. Cases of vestibular nystagmus are more irritable and more liable to vomit during or after the syringing. The central cases are irregular and the vestibular ones are regular. This is true because brain tumors develop slowly, and nature has an opportunity to adapt herself to the slowly increasing intracranial pressure up to a certain point. The left ear failed to react to cold syringing.

If she stood erect, with her feet together and her eyes closed, she fell to the right; if she tried to walk a straight line, she deviated to the right. While standing erect with her head turned to the left, she fell forward. In all positions except one she fell to the right of her head. Changing the direction of the quick component from one side to the other is indicative of a brain lesion. Nystagmus due to a brain tumor (central) is apt to increase in severity, while that due to a vestibular disease diminishes. For instance, a cerebellar abscess will suddenly increase the nystagmus. The changeable character of the nystagmus is indicative of a brain lesion. In this patient, at one time the nystagmus was rotatory, at another horizontal, and later vertical. Turning to the left produced nystagmus to the right for seventeen seconds, while during the turning she had strabismus.

When one labyrinth has been entirely destroyed the other becomes compensatory. Therefore, the caloric reaction is lost on one side, but it is present on the other, so that the turning test is normal. The finger test was negative. It may, however, change at any time to the abnormal and then back to normal again. At one time she falls to the right, but at another she may fall to the left. The galvanic test was negative. The corneæ were sensitive and the facial nerves normal.

Diagnosis.—Cerebellar tumor. It is possible that the tumor was farther forward, but it exerted pressure upon the cerebellum.

Case 19.—A man, age forty-five years, presented himself, July 26, 1911, for examination, with the following history: In 1898 his head was run over by a carriage, and the next day he had nausea, vomiting, vertigo, tinnitus and deafness. His hearing grew steadily worse, so that in two months he was entirely deaf in the right ear. In 1909 he thinks he had middle ear catarrh. Ever since the accident he has had, from time to time, a swelling, with pain, behind the right ear.

The examination of the hearing revealed the following: The right ear was totally deaf. With the left he heard a conversational voice at seven meters and the whispered voice at two to three meters. By Weber's test the sound was lateralized in the left ear. Rinné's test was positive for the left and infinitely minus for the right, as he heard the fork when held over the right mastoid only in the left ear. The bone and air conduction were both shortened for the left ear, although he heard forks C 1 to C 4.

Examination of the eyes showed an absence of spontaneous nystagmus. The right ear was then syringed and produced nystagmus to the left, which was normal. Turning to the left ten times produced nystagmus to the right for twenty seconds, and turning to the right ten times produced nystagmus to the left for fifteen seconds, which is normal. The finger test was normal after turning to the right, but after turning to the left the patient either struck the observer's finger part of the time or passed to the right of the observer's finger. (Bárány has seen a few cases which are atypical, and this case is atypical.)

This patient's deafness was due either directly or indirectly to the traumatism. If directly, it must have been due to a fracture (basal) or a hemorrhage or both. If indirectly, to atrophy or degeneration of the eighth nerve. The cochlear division of the eighth is more susceptible to toxins and trauma than the vestibular. This patient had the symptoms of a basal fracture for three weeks after the injury, so the cochlea or cochlear nerve must have been involved. Possibly there was also some degeneration of the vestibular nerve. This can be determined by the galvanic test of both ears simultaneously or by syringing both ears simultaneously. Syringing both ears simultaneously with cold water produced in one-half minute nystagmus to the right, showing that the right vestibular apparatus was less irritable than the left.

Diagnosis.—Basal fracture involving the right side in 1898, with subsequent atrophy of the eighth nerve on the right (cochlear division entirely and vestibular partly).

BIBLIOGRAPHY.

1. "Researches on the Rhythmic Nystagmus Reflected from the Vestibular Apparatus of the Ear and Its Accompanying Symptoms." Monatschrift für Ohrenheilkunde, 1906.

XXXVI.

SOME CONDITIONS ASSOCIATED WITH THE LOSS OF CEREBROSPINAL FLUID.*

By R. Bishop Canfield, M. D.,

Ann Arbor.

I wish to relate to this society the following cases, during observation upon which, either before or after operation, there appeared an escape of cerebrospinal fluid, and to draw some conclusions from them.

Case 1.—Mrs. Lizzie A., aged fifty-two years, Jackson, Michigan. Patient presented herself on account of a suppurative otitis media on the left side, with dizziness. During childhood she had had scarlet fever, since which time the ear has discharged at intervals. Four years ago she had a mastoid operation in San Francisco. The ear and mastoid healed after about one year's postoperative treatment. Patient remained well for some months, following which occasional suppuration has been noted. About three weeks ago swelling appeared over mastoid. Since this time she has been dizzy, staggers to the right, and has been frequently nauseated.

Examination of the left ear showed great destruction of the tympanic membrane; tympanum contained small quantity of somewhat foul smelling, purulent discharge. Easily bleeding granulation tissue noted around the oval window. Mastoid scar infected, red and swollen.

Hearing test.—Conversational voice, not heard. Weber to the right. Rinné negative. Schwabach heard in the right ear. C-1 not heard. C-4 not heard. Nystagmus of one degree to both sides.

Turning test.—Turning to the right, after-nystagmus of fifteen seconds. Turning to the left, after-nystagmus of fifteen seconds. Caloric test, negative. Fistula test, negative.

---

*Read before the American Laryngological, Rhinological and Otolaryngological Society, Washington, D. C., May 10, 1913.

Radical mastoid operation. Mastoid showed the effect of previous operation, pus and granulation tissue in the antrum, middle ear and attic, easily bleeding granulation tissue about the oval window. Footplate of the stapes in position, although very loose. Labyrinth not opened. Good recovery from the anesthetic.

For eight days after the operation patient showed a spontaneous rotatory nystagmus to the right, which gradually disappeared. During the after-dressings the stapes could not be seen. During this time there was an oozing of clear fluid from the oval window. This reappeared as fast as it could be mopped away. At several dressings it was observed long enough to make it certain that more fluid appeared than could have been contained in the labyrinth, consequently it was concluded that this must be cerebrospinal fluid. This escape of fluid persisted for about ten days, after which it ceased. Recovery uneventful.

Case 2.—Mrs. Polly R., age forty-six years. Patient was referred to the clinic of otolaryngology on account of a right suppurative middle ear disease. She had had measles forty-two years ago, since which time her ear had continued to discharge. About seventeen years ago had periodic attacks of dizziness. At this time several polyps were removed, which operation relieved her of her vertigo. For many years she has had pain on the right side of the head in the parietal and mastoid region. She suffers from occasional attacks of vertigo and feels like falling to the right. Two weeks ago had a severe chill.

Examination.—Conjunctivæ both sides anesthetic. Pharynx slightly anesthetic. Right ear, marked destruction of the tympanic membrane, tympanum contains foul smelling pus, mastoid slightly tender to pressure.

Hearing test.—Conversational voice, not heard. Weber to the left. Rinné negative. Schwabach heard in the opposite ear. C-1 not heard. C-4 not heard. Spontaneous nystagmus, none.

Turning test.—Turning to the left, after-nystagmus of twelve seconds. Turning to the right, after-nystagmus of twelve seconds. Caloric test negative. Fistula symptoms positive.

July 5, 1912. Radical mastoid and Hinsberg labyrinth operation. Superficial sclerosis. Deeper parts of the mastoid, antrum and middle ear filled with pus and cholesteatoma. No fistula through canal system noted. Stapes absent. Oval window covered by elastic membrane resembling scar. Upon opening the vestibule there was free hemorrhage; granulation tissue not seen.

July 17, 1912. Patient complains somewhat of dizziness. Horizontal and rotatory nystagmus to the left of one degree. Two days later nystagmus had disappeared.

July 28, 1912. Clear fluid escaping from the labyrinth. This fluid of sufficient quantity to preclude its formation in the labyrinth. Discharge persisted for about ten days.

August 24, 1912. Patient well.

Case 3.—Mrs. Sarah V., age forty-three years, Ann Arbor, Michigan. Patient presented herself in the clinic of otolaryngology with a history of having had a discharging ear since childhood. She has occasionally had severe pain over the left mastoid and left side of head, and has had occasional attacks of dizziness, during which she has not fallen, but has felt great discomfort. Examination of the left ear showed the deeper parts of the canal filled with foul-smelling crusts and pus, with some thickening of the anterior inferior canal wall and great destruction of the tympanic membrane. Shrapnell's membrane, posterior to the short process, was lacking; stapes and incus plainly seen through perforation. After considerable nonoperative treatment without improvement in her condition, a radical mastoid operation was advised. This was performed April 27, 1909. A tedious but satisfactory convalescence was established and the patient made a satisfactory recovery.

October 28, 1909. Since the radical mastoid operation there have been times when the ear would discharge somewhat. During the periods of discharge she felt perfectly well, but between attacks complained of some dizziness and pressure in the head. Examination of the ear at this time showed it to be completely epidermatized.

January 31, 1910. For several weeks the patient felt perfectly well and gained in weight; ear has remained dry. Recently she has had some dizziness and pain in the head and sense of discomfort in the left eye. There is a slight horizontal nystagmus of one degree to both right and left. From this time on the patient was treated occasionally in this and the neural clinic.

October 14, 1911. Nystagmus of one degree to both right and left. Turning to the right showed after-nystagmus of twenty-two seconds; turning to the left showed after-nystagmus of forty-five seconds, with marked dizziness. Irrigation of the left ear with cold water showed slight horizontal and rotatory nystagmus when eyes were turned to the opposite side, lasting a very few seconds, without dizziness. Irrigation of the right ear with cold water showed horizontal and rotatory nystagmus to the left, lasting one minute and fifty seconds, with marked dizziness.

October 17, 1911. Turning to the right gives an after-nystagmus of twenty seconds with marked vertigo. Turning to the left, no after-nystagmus and very slight vertigo. Irrigation of the left ear with cold water gives a slight horizontal and rotatory nystagmus to the opposite side. Patient stone deaf. Leucocytosis, 10,500.

November 1, 1911. The following operation was performed under ether: Old mastoid wound opened up, found perfectly epidermatized. Wound cavity discovered as at previous operation. As the operation progressed there was discovered a sac capable of holding probably one-half dram of material, with an opening into the tympanic end of the eustachian tube. This sac was curetted away. The cerebellum was uncovered and an attempt made to open the labyrinth from behind. On account of the escape of cerebrospinal fluid from a small circular opening in the dura, which was probably a large perilymphatic duct opening atypically into the subdural space, this stage of the operation was not permitted. The labyrinth, however, was opened widely from in front. As a result of the packing necessary to control the flow of the cerebrospinal fluid, the opening in the dura was much enlarged. One stitch was then taken in the dura. The patient made a good recovery from the anesthetic.

November 3, 1911. Following the operation the patient's subjective symptoms improved remarkably, and she said that she felt better than for a long time. She developed a slight nystagmus of one degree to the opposite side, which persisted for three or four days. On the second day following the operation the amount of cerebrospinal fluid lost increased so markedly that the bandages, pillow and upper part of the bed were soaked for three days. The patient developed a temperature, stiff neck, Kernig's, Babinski, edema of the retinæ and some opisthotonos. She became somewhat stuporous. However, a leucocytosis did not develop. It was apparent that the patient was rapidly growing worse and without some further operative interference would probably die shortly. On this account the following operation was done at midnight: The dura of a dog was secured and tamponed firmly against the cerebellum in such a way as to cover the exposed cerebellar tissue and compleetly close the opening, which at this time was about the size of the middle finger. Following this procedure the patient was put in bed with the head of the bed elevated. Following this operation the cerebrospinal fluid ceased to escape, and there was a marked improvement in the patient's subjective condition with decrease in pain and decrease in temperature.

November 7, 1911. Despite occasional attacks of severe occipital and right-sided headache, and the fact that her temperature ranges from 101 degrees to 104.6 degrees, the patient's subjective condition continues to improve. Today patient had a slight nose bleed. The wound was dressed and found perfectly clean, the dural graft was in position and there was no escape of cerebrospinal fluid. A Widal test was ordered and was negative.

November 9, 1911. For each succeeding day the highest temperature is lower than the day before, the highest for today being 102.6 degrees. Last night patient complained of pain in the epigastrium and fainted. An ice cap was placed over the heart and a turpentine stupe over the abdomen. The patient slept very little. She feels fairly well today. The wound was dressed and found perfectly clean. Although there was some discharge on the dressings, there was no evidence of the escape of cerebrospinal fluid, and the dural graft was in position. The white blood count was 6,500.

Then followed an uneventful convalescence.

Case 4.—Karl S., age twenty-three years, Kalamazoo, Michigan. Patient was referred to the clinic of otolaryngology on account of a discharging ear. He was well until six months ago, when he became rather suddenly dazed and stupid. He continued in this condition for about three weeks, without much fever or other symptoms than this mental terror. After this he made a gradual improvement and gained considerably in weight. After about two months he was able to look for work, although he did not take a position. About six weeks ago he took cold, at which time the ear began to pain. Pain in the ear and discomfort in the right side and back of head continued until this time. During this time has also complained of occasional severe frontal headache. A week ago the ear began to discharge a clear fluid. Five days ago complained of severe frontal headache in the evening, and on the following morning was found unconscious. He remained unconscious for twenty-four hours, and since then has been semiconscious and has complained of great pain in head and neck.

Examination.—He appears stuporous. Answers questions indifferently well and slowly. He knows his name, but not where he is. His neck is stiff. Double Kernig's and Babinsky. Patellar reflex absent. Pupils dilated. Nerve head and fundi slightly fluffy. Double facial paralysis, more marked on the left side. Leucocytosis, 28,000. Urine shows large quantity of albumin and many casts. Cerebrospinal fluid is purulent and contains many streptococci. Does not reduce Fehling's solution. Right ear concha is filled with perfectly clear fluid, deeper parts of canal contain pus. A clear fluid is seen escaping from the Rivinian segment. It pulsates rapidly and is apparently cerebrospinal fluid. Tympanic membrane is dark and retracted in the anterior portion and bulging in the posterior half. Mastoid not tender. Tongue dry. Buccal cavity and tongue covered with crusts. Teeth covered with sordes. Drainage of the basal cistern was immediately made according to the method of Haynes. When the dura was opened, cerebrospinal fluid escaped freely. This fluid contained large floccules of pus. The base of the brain was irrigated with warm normal salt solution during the operation. The patient recovered from the anesthetic in good condition, although respiration was very slow and deep, six per minute. It was noted that immediately after the lumbar puncture, which was done before the operation, the ear ceased to discharge. Two days later the patient died.

Autopsy showed a plastic basal meningitis with extension along the fissure of Sylvius. The apex of the right temporal bone was occupied by a very large pneumatic cell containing cerebrospinal fluid and pus. The perforation through the wall of this cell had permitted the entrance of infection to the subdural space and the escape of the cerebrospinal fluid externally into the pneumatic structure of the temporal bone, thence to the middle ear and external canal.

Case 5.—William S., aged forty-two years, Kalamazoo, Michigan. Patient was referred to this clinic on account of a carcinoma of the temporal bone which had been present for about a year. Examination showed an extensive squamous cell carcinoma, involving the external canal, middle ear and mastoid, and which had perforated through the mastoid cortex to involve the soft parts. The labyrinth did not react to the physiologic tests.

December 27, 1912. Radical mastoid operation was performed and a large mass of carcinoma removed, together with the auricle. Patient made a good recovery and the wound became clean.

February 5, 1913. On account of the appearance of a spontaneous nystagmus of the third degree to the diseased side and marked disturbance of the equilibrium, it was decided to resect the temporal bone. This was accordingly done. It had been the intention to remove the affected meninges and to do a dural transplantation, but inasmuch as it was discovered during the operation that the disease was too extensive, this transplantation was not made. During the operation and for some days afterwards there was a free discharge of cerebrospinal fluid. This, however, ceased and the patient made a good recovery. He was discharged to the care of his family physician to die a month or two later as a result of the recurrence of the growth.

Case 6.—Elizabeth M., age four years, Frankenmuth, Michigan. Patient was referred to the clinic of otolaryngology from the neural clinic with the following history: Nine months ago she fell on a toy boat, the mast of which was made of a knitting needle. The needle entered the orbit just below the eye. A piece of the needle two inches long was removed immediately and the wound healed. It did not affect the eyeball or vision apparently. The wound healed within a few days. After that she complained of headache, vomiting, more or less constant sleeplessness, and about four weeks after, an incision was made at the inner side of the eyeball, from which pus escaped. Six weeks after that Dr. Slack of Saginaw operated upon the orbit and removed another piece of steel about two inches long, which showed blunt and broken ends. The parents believe that the needle was broken off before the accident happened and that this part is the end of the needle, but this is not absolutely certain. The child remained well after this part was removed until about seventeen days ago, when she began to complain of headache, sharp and piercing in character. She vomited and was sleepless. No dizziness nor staggering was noted. When the trouble began seventeen days ago she complained of pain only in the left frontal region, but now the pain is on both sides. She vomits less frequently now than formerly, and has slight fever. Temperature has been moderate, from 99° to 100° and 101° or more in the evening. She has not had any cross eyes in the last seventeen days. There is no rigidity in the back of the neck. She is irritable to noises and light, but apparently not excessively so.

Neural examination: Patient is able to sit up, whines constantly, has an expression as if in pain. Says that she is in pain, but does not know where it is. She responds to questions, but evidently does not understand fully. Head: The pupils are equal and react to light and accommodation. There is no strabismus nor extraocular palsy. No tremor or atrophy of the tongue, no facial paralysis, no fever, no rigidity or retraction of the back of the neck. There is a small scar just outside the internal canthus of the left eye in the lower eyelid, slight ptosis. No Kernig or Babinsky. Chest and abdomen negative. Urine negative. Blood: Red, 5,125,000; white, 16,000; hemoglobin, 70 per cent.

December 10, 1910. Transferred to this clinic for examination and operation. Ear, nose and throat examination negative.

Operation.—Usual frontal sinus incision through the left eyebrow to the median line. Vertical incision upward two inches long in the median line, periosteum retracted, cortex negative. Frontal sinus opened and found negative. Entire frontal wall of frontal sinus removed. Dura uncovered one inch in diameter. Dura bulged strongly forward, marked pulsation. U-shaped flap, base upwards, one-half inch in diameter, followed promptly by small brain hernia. Brain deeply incised in all directions with negative result. Dural flap sutured with catgut. Skin incision sutured for primary union. Good recovery from ether. For a week following the operation the patient was in good condition, pain on the left side of the head slightly greater than before operation. Temperature normal.

December 23, 1910. Having been restless for the past two or three days, on this morning she suddenly lasped into semiconsciousness, from which she could be aroused with difficulty. Both optic discs were swollen.

Operation.—Original incision opened, dura found bulging strongly; area of bulging was incised and a large amount of creamy pus escaped. Following the escape of pus clear fluid escaped from the depths; no doubt cerebrospinal fluid. For ten days following the operation both pus and pulsating spinal fluid escaped at the time of dressing. The wound was dressed daily with a Whiting brain searcher.

January 16, 1911. For sixteen days the wound has been clean, and no cerebrospinal fluid has escaped; that is, the ventricular fistula has closed.

Operation.—Wound edges freshened and sutured. During this operation it was noted that the dura bulged forward through the opening in the skull the size of a small hazel nut. It was very elastic and thin, and there seemed to be some fluid behind it. On account of the absence of symptoms and the fact that the brain had been searched carefully on several occasions since bulging had taken place, the brain was not opened.

January 19, 1911. For two days following the operation the patient remained in good condition, but on the following day had a rise of temperature, became very irritable and showed a leucocytosis of 21,000. Her disposition changed suddenly and completely; she became irritable and showed violent fits of temper. She complained of severe headache on the left side of her head. On account of these symptoms the skin incision was reopened and the brain incised, brain searcher introduced and a second abscess discovered at a somewhat deeper level than that occupied by the first one. Immediately following the operation the patient's condition changed entirely and she became happy and contented. Her convalescence was uneventful, except for an attack of nasal coryza.

This case is of considerable interest because of the fact that the abscess was a double one and could be clearly demonstrated as such. Again, it is a very rare thing to have a deep brain abscess recover. It is also interesting to note the effect of the abscess on the child's disposition. When she was brought in for the second operation she was the picture of a very naughty child with a violent temper; she cried and struck at her nurse; but after the abscess was drained and immediately upon coming out from her anesthetic, she showed a very sweet and lovable disposition. Since this operation she has been perfectly content, happy and obedient. At the present time she has a slight divergent strabismus and a slight regularity in her gait.

The method of conducting the after-treatment of this brain abscess may be worthy of mention. The abscess was neither packed nor irrigated, but was investigated daily by means of the Whiting brain searcher, an instrument which permits one to examine thoroughly every part of the abscess cavity, wipe it out gently and in this way by means of reflected light to control satisfactorily the healing process.

Case 7.—Glenn S., age eighteen years, Paw Paw, Michigan. Patient was referred to the clinic of otolaryngology on account of discharging wound in forehead and his mental condition. Patient well and strong until the present illness. Two months ago he had a head cold with free discharge of matter from the nose. During this time complained of great headache and sleepiness. Patient would answer questions, but would drop off to sleep during conversation. Slept most of the time. This condition of drowsiness lasted for two or three days. Patient not in bed, but apparently weak. Felt better and walked four miles. Then took an eighteen mile drive to visit his brother. During his visit complained of headache, chiefly in the left frontal region. At times would appear normal and then again stupid. Nose ceased to discharge. Remained home for about three weeks, gradually getting weaker and complaining continually about the pain in his head. His father noticed that he was somewhat confused in his speech. He frequently said that he was feeling "bigger," no doubt meaning that he felt better. He began to complain that he was seeing double, and the father noted that the left eye turned in toward the nose. He was taken to a hospital, where an operation was performed. After the operation he made some improvement, his eyes became straight and his mental condition improved. Three weeks before his admission into this hospital he was again anesthetized, and an intranasal operation was performed for the purpose of securing drainage from the left frontal sinus. He continued to improve and was finally sent to his home physician for observation. During his residence in his home hospital he was not dressed by his physician, but was cared for by the nurse, from which it may be thought that his condition was not considered serious. After returning to his home the wound in his forehead did not heal, but continued to discharge foul pus. His mental condition did not arouse comment. On the night previous to his coming under my observation he began to show mental derangement; for example, when his father attempted to give him an enema, the patient placed the tube in his own ear. Later in the evening he becam stupid, his answers became delayed, and he again spoke of feeling "bigger" instead of better.

When he was brought to the University Hospital he was somewhat emaciated, but was strong enough to assume the active position in bed and to turn towards the examiner without assistance. He showed no paralysis. The knee jerk was slightly increased on the right side, and there was slight evidence of a Babinski's sign on that side. He also showed a double optic papillitis, more marked on the left side. His breath was foul, and the same foul odor was noted in the perspiration. He knew his name, his home and where he was, but in other matters his memory was defective. He answered questions intelligently but slowly, and always in complete sentences. If asked if he had pain, he replied, "I have no pain," etc. As soon as he had answered he would close his eyes and sink into a sort of stupor.

A sinus was present in the center of the left eyebrow, through which foul pus escaped. A probe passed gently into the sinus discovered no bone. On account of the location of the suppurative process and the patient's mental condition, a diagnosis of probable left frontal lobe abscess was made and an immediate operation advised.

May 25, 1910. Operation.—A U-shaped flap was turned up from the left forehead. The anterior wall of the left frontal sinus was absent, and the frontal sinus was found filled with foul pus and prolapsed brain tissue. An opening through the posterior wall of the frontal sinus, about one-half inch in diameter, was discovered. Over this area the dura was gangrenous, while the bone in the neighborhood was carious. This condition rendered the formation of an osteoplastic flap impossible. The inner table was then removed until healthy dura was uncovered in all directions. To do this it was necessary to uncover an area about one and one-half inches in diameter. A sinus was discovered through the dura at a point about one-half inch above the center of the eyebrow. Through this sinus pus escaped. On account of the condition of the dura, a dural flap was impossible; therefore the brain was freely incised to permit the escape of indescribably foul pus, the odor of which must be experienced in order to be appreciated. This pus continued to escape for about fifteen minutes. During this time a very small amount of anesthetic was needed, and the pulse increased about twenty beats per minute. The right frontal sinus was opened and found to be filled with foul pus and necrotic tissue. The frontal sinuses were packed with iodoform gauze, the brain cavity not being packed. No sutures were taken. The patient regained consciousness on the table and was returned to bed feeling better than before the operation.

At the second dressing about one and one-half ounces of cerebrospinal fluid escaped from the depths of the wound, and from them on daily for a week cerebrospinal fluid was seen to escape, at the end of which time the ventricular fistula healed. He died three weeks after the operation without symptoms of meningitis.

Case 8.—Mr. B., age thirty-five years, Detroit, Michigan. Patient was referred to the clinic of otolaryngology from the surgical clinic, where he was being treated for discharging sinus near the median line in the left frontal region. Present trouble began nine months ago, at which time he struck his head on the fitting of a steam pipe. Later had pain and spontaneous rupture of the skin, with free discharge of pus. At this time the skin was freely incised at about the midpoint of the left frontal sinus, and since that time he has had more or less persistent purulent discharge. Patient states that nine years previous to this he had an accident while in a lumber camp, which resulted in unconsciousness for forty-seven days and necessitated trephining. Present examination showed the left eye missing (due to the accident nine years ago above mentioned). Deep crater-like scar over the midpoint of the left frontal sinus, at which point patient had been trephined, and median to this is a small granulating sinus from which escapes thick pus. The throat is negative except for chronic pharyngitis. Nose shows small sharp spur low down on the right side, high deviation and small dislocation of the cartilage on the left side. No pus noted in nose. Transillumination showed right frontal sinus to transmit light freely; less light is transmitted on the left side. Ethmoid regions on both sides transmit light freely. Transillumination of the antra normal, light reflexes and sclera being obtained on both sides. Radiograph shows shadow inside the cranium at the level of the frontal sinus. Physical examination was negative.

October 10, 1912. Operation.—Incision through the left eyebrow with vertical incision upward to neighborhood of hair line. Removal of the anterior table showed frontal sinus filled with pus and granulation tissue. Brain uncovered for a considerable area corresponding to part of posterior wall of frontal sinus. Depressed fracture the size of thumb nail. Trephine opening over the frontal lobe lateral to and above frontal sinus, showing considerable new bone formation. Curettage of frontal sinus, removal of the entire anterior wall together with roof of orbit, discloses necrotic bone below area of uncovered dura, removal of which uncovered large area of the left frontal lobe and part of the right frontal lobe. Old rupture of dura noted over halfmoon-shaped area one-fourth inch in diameter, situated just below and in front of trephine opening. This rupture covered with very thin scar, which was torn during the operation, allowing the escape of a small quantity of cerebrospinal fluid. Wound irrigated and packed.

At the second dressing cerebrospinal fluid was seen oozing from the perforation in the dura near the outer angle of the wound. Wound otherwise clean and granulating. This escape of cerebrospinal fluid continued for four days, after which it ceased. Patient then passed through an uneventful recovery.

Case 9.—Mr. Van F., age fifty-two years. Patient was referred to the clinic of otolaryngology from the clinic of neurology, where he is being treated for right sided hemiplegia. Seven months ago patient fell fifteen feet, striking on his forehead. He did not lose consciousness, but walked to the house. Had a violent nose bleed, which lasted for two hours, at which time his doctor sewed up the wound in his forehead. About two weeks later he began to have difficulty in walking. This seemed to be due to weakness in his right leg. Later right arm became weaker, and he soon began to have difficulty in talking. Found that he could not remember the words he wished to use. Whenever he sits up, clear fluid to the extent of two ounces escapes from the left nostril, at which time he has left-sided headache. On several occasions, during which cerebrospinal fluid has been lost, he has become unconscious.

Neural examination (Dr. Camp): Patient's station with eyes open is fairly good, swaying slightly; but with eyes closed the swaying is very marked. The gait shows ataxia in the right leg, so great that he can only walk three or four steps without support. There is a slight right sided facial palsy, shown by a weakness in wrinkling the forehead on the right side, a slight aperture in the right eye when eyes are closed, and a lack of firmness in the right cheek when both are inflated. On protrusion the tongue goes slightly to the right. There is a very marked weakness in all movements of both right arm and leg. Dynamometer test with the right hand is 40, with the left 115. No ataxia is noted when the patient makes circles with the left leg, but with the right the movements are jerky and faulty. Some ataxia is shown in the right arm when the patient is asked to touch the end of the nose with forefinger. There is a subjective numbness throughout the whole right side of the body, but no anesthesia to touch can be demonstrated. Pain conduction is apparently delayed throughout the right side, as the patient says that he feels a pin point more keenly on the left side. There is no difference felt in the surface temperature between the two sides of the body. Reflexes: The pupils are equal and react to light and accommodation equally but rather sluggishly. The extraocular movements are normal. The biceps, triceps, kneejerks, and Achilles reflexes are all active on both sides, but more so on the right. The abdominal and cremasteric reflexes are present and about equal on the two sides. Well marked ankle clonus and a Babinski reflex are present on the right side, but not on the left.

Measurements: Upper arm at corresponding points, right, 24 centimeters; left, 27 centimeters; forearm, right, 22½ centimeters; left, 24 centimeters; thigh, right, 35 centimeters; left, 37 centimeters; calf, right, 27 centimeters; left, 29 centimeters.

Hearing test shows air conduction to be better than bone on both sides. The sense of smell is lost in the left nostril and diminished in the right, where he smells but cannot recognize accurately. He recognizes salt on both sides of the tongue, but does not recognize sweet or sour on either side.

The physical examination is negative, except for a marked depression, the size of a quarter, above the left eye, which is the site of the trephine done the night of the accident. There is some tenderness to pressure here, but no pulsation. The examination of the blood and urine is negative. Three days after his admission the eyes were examined in the ophthalmologic clinic and the following report received: Edematous retina. No hemorrhages nor papillitis. Convergence limited in the left eye. Upon sitting erect there was a loss of from one to one and one-half ounces of cerebrospinal fluid (Dr. Canfield). An exploratory operation was therefore decided upon.

July 31, 1909. Operation.—In the left frontal region U-shaped incision made and flap turned down, dissected away from adhesions about the old operative field and from the dura, which disclosed an opening in the bone one by one and one-fourth inches, from which perforation in dura was plainly visible, this perforation being three-eighths by one-half inch. Probe passed through this opening came in contact with brain; found brain collapsed so markedly that anterior surface of the frontal lobes lay one and one-half inches from dura. Dura incised and plastic done, closing the orifice by means of silkworm gut. Flap suture in place. The second day the stitches were removed and collodion dressing applied.

August 10, 1909. Patient in good condition, wound entirely healed, patient feels stronger, has strong grip in right hand, has had no more headaches and claims to be regaining control of right leg. About one month later patient resumed his duties as mail carrier, apparently perfectly recovered, although he was showing some signs of beginning general paresis (specific).

Case 10.—Mr. John F., age forty-nine years, Pontiac, Michigan. Patient was transferred to the clinic of otolaryngology from the surgical clinic, where he had been operated upon twice for alveolar sarcoma of the neck.

Examination at this time showed tumor mass the size of a large fist, presenting in the right frontal region. This mass is fairly dense with soft areas. It involves the right upper eyelid, displaces the right eye markedly to the right and forward and downward, extends over the median line. Some edema of both left eyelids, also marked edema of the right lower eyelid. Shows a scar in the right upper eyelid, also a sinus in the right frontal region from which pus escapes. Mass extends upwards two-thirds of way to hair line, not attached to the skin, except at the two points mentioned. Nose shows the right nostril completely occluded by tumor mass, which is not attached either to the septum or the lateral wall of the nose anteriorly. Free purulent discharge from the nose. Neck shows several scars on the right side. Dense mass, the size of a large English walnut, not attached to the skin, lies under the anterior margin of the right sternomastoid. Left side of neck shows scars.

January 16, 1913. Operation.—Enucleation of a mass lying attached to the sternomastoid. Removal of one lobe of the submaxillary salivary gland which was lying in relationship with tumor.

January 18, 1913. Operation.—Under ether. Horizontal incision beginning at the outer angle of the left eyebrow and extending across to midpoint of the right eyebrow, and vertical incision in median line. Retraction of the soft tissues permitted the removal of a large mass of malignant growth. After this had been removed it was discovered that the outer table was lacking over the entire right half of the frontal bone and part of the left frontal bone. Tumor mass was scraped out of a large frontal sinus. Right frontal lobe occupied by the tumor to a considerable depth. Profuse hemorrhage, and operation was stopped at this point. Wound packed and incision sutured.

January 19, 1913. Wound dressed, escape of a considerable amount of clear cerebrospinal fluid from the deeper portion of the tumor; that is, from the lateral ventricle.

January 30, 1913. Daily escape of cerebrospinal fluid for four days, following which the escape ceased and the wound cleared up.

February 28, 1913. Patient discharged to the care of his home physician with evidence of tumor growth.

Case 11.—Patient, Alexander H., presented by courtesy of Dr. A. M. Barrett of the State Psycopathic Hospital, Ann Arbor, Michigan. Patient admitted August 20, 1906, suffering apparently from dementia. He appeared stupid and indolent, but responded accurately to questions. He is partially blind. Speech is thick, slurring, explosive and ataxic. During the past ten days patient has been noted holding his handkerchief over his nose, and when this was investigated it was noted that he had a serous discharge from the nose at the rate of about one drop per second. This fluid was found to contain a small percentage of albumin, fibrin and leucocytes. Cerebrospinal fluid was suspected. Death later from natural causes.

Autopsy.—Head Section: The surface vessels were enlarged. Upon removal of the brain the base of the skull showed marked signs of increased pressure, the cribriform plate being eroded and the brain substance protruding into the nasal cavity. The cella tursica is much enlarged, round and unusually deep. The middle cerebral fossæ are much deepened, and the floor in some places is eroded, due to pressure. The posterior cerebral fossa shows marked signs of pressure, being extremely irregular. On removing the cerebellum the inferior surface is seen to be unusually full, especially on the right side. The temporal and occipital regions of the left side of the brain are hollowed out as though from pressure. The aqueduct of Sylvius is dilated and small, dark granular mass protruding slightly from it. On the section through the upper part of the pons a small tumor mass 8 mm. by 9 mm. in diameter, is seen filling the right dilated aqueduct of Sylvius and greatly compressing the brachium of the same side. The lower portion of the sectioned pons shows the tumor mass completely filling the greatly dilated aqueduct. On the horizontal section through the vermiform process of the cerebellum, extending down to the fourth ventricle, the tumor mass, 2.5 cm. by 3.5 cm. in diameter, is seen filling the widely dilated ventricle, producing greater pressure upon the pons and medulla on one side and the cerebellar tissue on the other. The tumor is reddish gray in color, choroidal in type, in its arrangement looking like innumerable small vessels closely crowded together. Cranial nerves not well preserved, the infundibulum is greatly dilated, being 12 mm. in diameter, and the transverse fibers of the chiasm are much less prominent than normal. Longitudinal section through the middle of the thalamus shows the lateral ventricles greatly dilated, especially on the right side, where the brain substance is seen bulging up in the posterior horn. The white and gray substance is greatly narrowed in all regions. Considerable injection of the fine vessels is seen. The ependyma of the lateral ventricles is coarse, granular and pinkish in color. The ependyma and brain tissue of the posterior horn on the right side seems to lie in folds. The pineal gland is cystic.

Sections from the tumor show it to be composed of massed loops and sections of tubes of cuboidal epithelial cells resembling proliferated choroid plexus tissue. In places there are cysts and masses of coagulated material. The tumor is evidently an epithelial tumor, springing from the choroid plexus of the fourth ventricle, and might be called an angioma or papillary epithelioma. The tumor by its location had caused an internal hydrocephalus which had produced a softening of the bones in the region of the base of the frontal lobes, and this explains the discharge of cerebrospinal fluid.

Cases Nos. 1, 2 and 3 are cases of labyrinth involvement occurring during a chronic suppurative otitis media. In case No. 1 the labyrinth was not opened at the time of operation, although failure to find the stapes after operation, together with the loss of cerebrospinal fluid daily for several days, are sufficient evidence of the fact that it had been opened.

In cases Nos. 2 and 3 the labyrinth was exenterated. Here are two cases in which the suppurative cavity was brought into communication with the subdural space without setting up a meningitis.

Case No. 3 is interesting in that it demonstrated the condition of meningismus into which a patient may lapse as a result of excessive loss of cerebrospinal fluid, and that a heteroplastic dural graft was successfully made for the relief of the condition.

Case No. 4 is interesting in that the patient showed evidence of meningeal involvement long before his attention was drawn to the ear condition, and also in that the avenue of infection of the meninges and the avenue of escape of the cerebrospinal fluid through the pneumatic structures of the apex of the temporal bone could be made out

Case No. 5 is another case in which free communication between the subdural space and the suppurating cavity did not cause meningitis.

Cases Nos. 6 and 10, that is, the case of double frontal lobe abscesses, and the case of sarcoma of the brain, show how easily sudden decrease in cerebral pressure may cause rupture of the lateral ventricles into the abscess cavity without infecting the ventricle.

Cases Nos. 7 and 8 again are illustrative of the possibility of bringing the subdural space and an abscess cavity into communication freely without setting up a meningitis.

Case No. 9 is most interesting, as it demonstrates that hemiplegia may result from loss of cerebrospinal fluid, and that function may be restored by stopping this escape.

Case No. 11 (psyco.) demonstrates that the subdural space or base of the brain may remain in free communication for an indefinite period with the nasopharynx without establishing a meningitis.

XXXVII.

ODD CASES OF NASAL DEFLECTION WITH SUGGESTION AS TO TREATMENT OF NASAL ADHESIONS.

By Otis H. Maclay, B. S., M. D.,

Chicago.

The ordinary operation is always found to be interesting and instructive, but the slight or extensive departures from the original line of work found necessary by exceptional cases is, I believe, even more interesting, owing, first, to the original technic that must be developed, and, secondarily, to the peculiar formations present. It is with the hope of satisfactorily describing some of the oddities found in nasal deflections and also the technic employed in correcting the same that I have ventured this article. I believe in about all of the very decided and very irregular angular deflections that trauma in some form or other is the etiologic factor, differing in this respect from those that are of a more regular curve, although these may be deflections of extreme degrees. As an aid in describing these irregularities, I shall first briefly mention the anatomy of the nasal septum and the relation to it of the perichondrium and periosteum.

The bony septum is composed in the main of the perpendicular plate of the ethmoid above, the vomer posterior and below, resting upon the nasal crest of the superior maxilla, and the anterior portion of the superior maxillary crest, or the socalled incisor crest. This anterior extension of the vomer, the superior maxillary crest and incisor crest, by their blending, form the lower portion of the septum, and are commonly spoken of as the "ridge." Placed in the space left by the articulations of the bony septum we find the triangular cartilage. The coverings of the cartilage and bone form separate envelopes for each part. The perichondrium of the triangular cartilage, as it approaches the ridge, passes under the cartilage to the opposite side of the septum, completely enclosing the cartilage. Similarly, the periosteum of the ridge passes passes over the bony crest to the other side of the septum, thus forming a complete envelope for the bone. These separate envelopes are closely united to each other at junction of cartilage and bone, and of course to the structures that they invest. This means with the ordinary deflection, which generally consists of cartilage and bone, that at least two separate envelopes must be opened before a complete removal of the deflection is possible. After having separated the necessary amount of perichondrium covering the triangular cartilage, and carrying the dissection to the covering of the bony ridge, we find we are unable to continue lower with blunt dissection without tearing the flap, since the area is reached where the perichondrium is turning under the cartilage and the periosteum over the ridge, going into the other side of the nose, and so at this point, the cartilage having been removed, it becomes necessary that the periosteum along the top of the ridge be incised and dissected from the bone down to the nasal floor. The union now of the periosteal layer below with the perichondrial layer above gives a large flap, extending from the desired line above to the floor, and when held by a retractor pulling laterally gives the appearance of a tent lying on its side, with the retractor acting as the crossbar at the top. Within these large flaps the bone can be readily seen and removed, either with chisel or bone biting forceps.

In order to demonstrate these points I have dissected a few septums. By examining them you will be able to see the relations of the various parts of the nasal septum to each other. These specimens, however, are used merely to aid the description, since they are not in most instances sufficiently irregular to warrant operative interference. With one exception the flaps have been dissected, not as done in the operation, but in order to demonstrate most clearly the various structures present. In specimen III an anterior view is given of the incision used and the freed flaps as seen while operating, with the exception of the right anterior inferior portion, where a fairly firm union is left as the cartilage rests upon the vomer. This shows how readily a tear could result if, without first freeing the flap at this point, the solid parts were removed. Specimen IV shows a portion of the perpendicular plate of the ethmoid placed for forward, and extending downward to within one centimeter of the anterior extension of the vomer, completely overlapped, however, on the right side by the cartilage of the septum.

In some of the specimens that portion of the cartilage overlapping the vomer is shown. It is very essential, as you can see, that a piece of cartilage so located must be removed before the bony covering can be freed. After its removal the covered vomer comes into view, and an incision is made through the periosteum, formerly covered by the overlapping cartilage, to the bone, and the dissection carried downward as far as desired.

Among the most common irregularities met are those where the cartilage is split, but lying in its own plane. There may be several pieces of cartilage, each with its own envelope, and consequently each piece must be freed of its perichondrium before removal. Again, the cartilage may be so broken and the deflection so extreme that the septum presses against the outer wall of the nose, with the split some distance posteriorly. A case with a vertical and horizontal deflection illustrates this. The extreme of deflection was vertically placed, about one inch inside the nose, but the bend extended horizontally the length of the cartilage. At the greatest point of deflection (vertical) the anterior piece of cartilage was found completely separated from the posterior portion, the latter part having its own envelope, which consequently was opened and bluntly freed. In this case the anterior piece of cartilage was first removed and then, having more room and clearer view, the posterior dissection was completed.

An irregularity that has required considerable care in my hands is one where the cartilage, extremely deflected, bends forward well into the opening of the nose. Here we have blending of the skin and mucous membrane, with the mucous membrane very much thickened. We wish to keep our incision well inside the nose as an aid in healing, and also to prevent the pain felt by incising the skin. Consequently we place our incision as far inside as possible, and work the anterior covering of the deflection forward. This is sometimes quite difficult, owing to the thick fibrous tissue present. The mucoperichondrial covering of the concave side in these deflections is often extremely thin, owing to the constant removing of crusts that form in the hollow of the cavity. Under these conditions, if the convex flap is torn in its freeing, great care must be exercised on the concave side, or a perforation might result.

Ordinarily the cartilage and bone occupy about the same relative space as the septum, but we find in exceptional cases a decided variation in the proportion of the two, and also in the thickness of the structures. In one case with a deflection extending posteriorly to a point opposite the middle of the middle turbinate, I was surprised to find the entire deviation consisting of the triangular cartilage, which was also very much thickened, measuring at the posterior portion at least one-fourth of an inch in width. Another case demonstrating the irregularity in amount of bone and cartilage showed the septum having a small anterior portion of cartilage, extending backwards for about one inch from the nasal opening, and the remaining portion of the septum being composed of perpendicular plates of ethmoid and vomer. This is the only one of my cases that has had the bone extending so far forward, although other cases have been reported.

In two cases, each having a moderate posterior deflection, but with a large, sharply pointed spur on the deflection, which extended well over to the outer wall of the nose, I removed both not only to give a better nasal condition, but because of the annoying tinnitus aurium present. The noise in the ears of both cases disappeared very shortly following the operation. Although appreciating the fact that there are many conditions producing such a disturbance, I mention this because of the result and not with the idea that tinnitus aurium is even likely to be benefited by such a simple remedy in all cases.

Among the various complications found in the septum work is a union between the outer and inner walls of the nose. This, when freed, results in two raw surfaces, with a tendency during the healing period of again uniting, since it is not uncommon to have considerable swelling for two or three days following the removal of the packing. In order to avoid such an unpleasant complication, I treat any denuded surfaces with thirty per cent silver nitrate immediately after removing the gauze. This is rubbed over the area; leaving the albuminate of silver for a covering eliminates the raw surface and consequently prevents later adhesions. I have employed this solution for several years, not merely for the septal cases described, but have used it wherever I wished to prevent adhesions in the nose. The other methods, such as packing and the actual cautery, caused a decided reactionary swelling, and often the same condition recurs. With the actual cautery we have the reactionary swelling not only from the burning, but also from the congestion arising after the effect of the cocain has disappeared. I have been much pleased with the results from the strong nitrate solution, and take this opportunity of describing its application.

After cocainizing, the adhesion is broken up and then the denuded area rubbed over with thirty per cent silver nitrate, which is applied with cotton on the end of an applicator, care being taken not to have the cotton more than dampened with the solution, thus limiting its application to the region desired. This can be treated within two or three days again, but without cocainizing, simply applying a small amount of the silver nitrate. At this time considerable slough will be present, but this should not be disturbed until it comes away without bleeding, thereby insuring against a new raw surface. If bleeding occurs, however, the solution should be applied to the area involved.

Another condition of the septum that is out of the ordinary is where the anterior end of the cartilage is dislocated to one side, and almost protrudes from the nose, with the remaining portion of the deflection (the larger part) extending into the opposite nostril; that is, a horizontal "S." I believe in this class of cases, if the conditions are suitable, that the best results are obtained if two incisions are used. First the extreme anterior deflection is removed, and, the incision being far forward, sutured with fine silk. Then on the opposite side an ordinary submucous dissection is done. This can be done if there is a sufficient amount of cartilage behind that first removed, which is not too greatly bent to the side of main deflection. As the bend of septum passes from the anterior piece which lies obliquely across the right nares, let us say, to the left nose, there is often a small piece of cartilage, one-quarter to one-half inch in length, that remains fairly well set in the median line. In the first operation remove cartilage to the anterior end of this medianly set portion and begin on the other side behind this same part, so that we have a large undisturbed portion of the cartilage of the septum left for support of the anterior end of the nose. This should prevent any later tendency of the tip from bending downwards, due to cicatricial contraction of the anterior operated area. As I have stated before, I have never seen this condition result, but believe the method just described a safe and conservative one. It gives a good result and saves tissue, and in all cases that should be our aim.

In one case the anterior inferior portion of the septum showed an old perforation from typhoid, about the size of a dime, with a marked left-sided deflection extending posteriorly, involving both bone and cartilage. The incision was placed sufficiently behind the perforation so that the opening in the septum was surrounded by cartilage. This was done to insure a firm anterior attachment for the freed mucoperichondrium, preventing consequently any tendency of the membranous septum from flapping upon blowing the nose. The greatest difficulty experienced in this case was due to the field of operation being set so far inside the nose, which necessitated considerable time in elevating the flaps, in order to keep the cartilage at the posterior portion of the perforation intact.

The cases described show to some extent the peculiarities met in this line of work, and also that they can be safely corrected and a good result obtained if sufficient care is exercised on the part of the operator.

XXXVIII.

THE OPERATIVE FINDINGS AND RESULTS IN MASTOIDITIS, ACUTE AND CHRONIC.

By J. M. Ingersoll, A. M., M. D.,

Cleveland.

It is the purpose of this paper to describe briefly the symptoms, operative findings and results in a few cases of mastoiditis. Naturally such a paper will contain little or nothing which is strikingly new, but the consideration of the symptoms and operative findings may help us to recognize similar cases early and by prompt operation prevent serious complications.

Case 1.—A boy, one year old. He had had a coryza followed by a purulent discharge from the right ear for five weeks. The temperature was normal, but the discharge from the ear was profuse, and the posterior wall of the auditory canal was bulging. On account of the profuse discharge and bulging canal wall, a mastoid operation was done and a large antrum filled with pus was opened. The discharge from the ear ceased in a few days, and the mastoid incision was entirely healed in two weeks. The hearing in this ear is now normal. This case illustrates the well-known fact that a profuse purulent discharge from the ear, continuing for more than four weeks, is an almost certain indication of infection of the mastoid antrum. If, in addition to the profuse discharge, there is some swelling of the posterior or superior membranous canal wall, the diagnosis of mastoiditis is positive, for the swelling is caused by the inflammation in the mastoid cells situated between the antrum and the auditory canal.

Case 2.—Male, thirty-five years old. Acute suppurative otitis for six weeks. Profuse purulent discharge with slight bulging of the posterior canal wall and tenderness over the tip of the mastoid. Temperature normal. Operation. The mastoid cells had practically all been destroyed, making one large cavity extending down to the tip, filled with granulation tissue and pus. The whole tip of the mastoid was necrotic and was removed as a sequestrum, The recovery was uneventful and complete, with normal hearing. This case shows how rapidly the whole mastoid may become inflamed and even partially necrotic without causing very marked symptoms. This patient had remained at home only two days at the beginning of the acute infection in the ear, and had not consulted any physician until the day before the operation. The tenderness over the tip of the mastoid indicated an infection in the cells there, but one would naturally expect more evident indications of so extensive an involvement of the bone.

Case 3.—Male, twenty-one years old. Profuse purulent discharge from left ear for six weeks. Granulation tissue projecting through the perforation in the drum membrane. Some swelling and tenderness over the mastoid bone. Temperature 99.8°. Operation. All the mastoid cells were filled with pus and granulation tissue. The sigmoid sinus had been exposed by necrosis and was covered with granulations. There was a small extradural abscess just above the sinus. Recovery was complete in three weeks.

This case also shows how extensive the destruction of the mastoid may be without causing very decided symptoms. The mastoiditis was self-evident, but there were no symptoms indicating brain abscess or any involvement of the sinus.

Case 4.—Male, forty years old. Purulent discharge from the ear for seven weeks following an attack of tonsillitis. Temperature 99.6°. The whole membranous canal was inflamed and tender, and there was considerable swelling above and anterior to the ear, extending forward as far as the orbit. Posterior to the ear there was very little tenderness or swelling.

The discharge from the ear had been gradually decreasing, and it was thought that perhaps the swelling around the ear was due to the external otitis, so that the patient was kept under observation for a few days. Then tenderness over the antrum became manifest and the mastoid was opened. The mastoid cells extended well forward into the posterior root of the zygoma and posteriorly back and around the sinus. They were all filled with granulation tissue and pus, and the cells posterior to the sinus had broken down, causing an extradural abscess. The wound was packed wide open. On account of its size it filled in slowly, so that it was necessary to dress it for eight weeks. It healed, however, without much depression and the hearing was normal.

In this case the swelling above and anterior to the ear and in the superior and anterior part of the canal wall was due to the infection in the cells which extended forward into the zygoma, and not to an external otitis. It would have been better to have operated when the patient was first seen.

Case 5.—Girl, three years old. She had had a severe coryza for four days, and was seen a few hours after she first began to complain of pain in the right ear. The drum membrane was red and bulging, and an incision was made in the posterior inferior quadrant. There was a free serous discharge which became purulent in two days. The temperature ranged between 101° and 103°. Twelve days after the incision in the drum membrane there was some swelling in the canal wall posteriorly, and the sternocleidomastoid muscle was slightly tense, with some fixation of the tissue over the tip of the mastoid. The latter two symptoms indicated that the cells in the tip of the mastoid were infected, and the mastoid was opened. The temperature came down to normal in three days, and for twelve days everything seemed to be progressing favorably. Then the temperature went up to 102° and a little granulation tissue appeared on the posterior canal wall. Under this tissue necrotic bone could be felt and a second operation was done at once. The posterior canal wall, down to the margin of the annulus tympanicus, the whole tip of the mastoid and part of the covering of the sinus, were removed, as there was some necrosis in all of these structures. Recovery after the second operation was complete in four weeks.

In this case the infection involved the mastoid, in spite of a free and early incision in the drum membrane, and although the first mastoid operation was as thorough and complete as the indications seemed to warrant, it became necessary to do a modified radical operation. The necrosis which developed after the first operation was probably due to the severity of the infection (streptococcus) and the low resistance of the patient.

Case 6.—Male, four years old. Chronic foul suppuration in left ear since infancy; middle ear filled with granulations. No tenderness nor swelling over the mastoid. Temperature normal.

Radical mastoid operation. A sequestrum containing most of the mastoid cells and the outer covering of the sinus was removed. The roof of the middle ear and antrum was necrotic, exposing the dura, which was covered with granulations. Because the sinus and dura were exposed, the inferior part of the mastoid incision was left open and packed. The ear was dry and healing was complete at the end of two months.

In this case the chronic suppuration had exposed the dura and the sinus, and the wonder is that the child had not died of meningitis or sinus infection.

Case 7.—Boy, five years old. Profuse, foul, purulent discharge from left ear for fourteen months, following scarlet fever. Middle ear filled with granulations. Temperature 105°. Restlessness, nausea and semiconsciousness, made the diagnosis of meningitis almost certain and the prognosis very unfavorable.

A radical mastoid was done a few hours after the boy was admitted to the hospital. The roof of the middle ear and antrum was necrotic. The dura was inflamed and tense, and so it was uncovered over an area about three centimeters in diameter and incised. There was a little serous discharge through the incision. The day after the operation the boy was slightly better, but on the second day he was worse and died on the third day. No autopsy was permitted.

If this boy's ear had been operated upon before the meningeal infection had taken place his life would probably have been saved.

Case 8.—Female, thirty years old. Chronic bilateral suppurative otitis media. Facial paralysis on the left side, dizziness, nausea, headache and spontaneous nystagmus to the right. After turning to the left the nystagmus to the right was decidedly increased for forty-three seconds. Turning to the right, nystagmus to the left for twenty seconds. Caloric reaction present in the left ear, but sluggish. Hearing (acoumeter) left ear, one centimeter; right ear, eighteen centimeters. Fistula symptoms absent. Temperature 99.2°. From the above symptoms the diagnosis of paralabyrinthitis, with some involvement of the facial nerve, was made and a radical mastoid operation done. The sigmoid sinus was covered by such a thin bony cortex that it could be seen when the periostium had been removed. It was so far forward that it was necessary to expose the sinus in order to get working space.

In spite of the negative symptoms a careful search was made for a labyrinthine fistula, but none was found.

After the operation the facial paralysis improved, the spontaneous nystagmus decreased and the temperature went down to normal. On the tenth day the temperature went up to 102°, there was considerable headache and necrotic bone could be felt in the region of the horizontal semicircular canal. The mastoid incision was opened, and because a fistula in the horizontal canal was found, a labyrinth operation was done. There was some necrosis of the bone over the facial nerve, and a small sequestrum was removed, leaving the facial nerve exposed. At the end of three months this ear was dry, but there was still some facial paralysis.

The right mastoid now began to be sensitive, and the patient had some nausea and headache. There was a perforation in the posterior inferior part of the drum membrane, through which granulation tissue covering the promontory could be seen. The rest of the membrane was in fairly good condition, and there seemed to be no necrosis of the ossicles.

On account of the mastoid tenderness, headache and nausea, it seemed necessary to operate upon this ear also, in spite of the fact that the left ear was absolutely deaf and that the hearing in this ear might be made less acute. A modified radical mastoid was done. The mastoid antrum was filled by a cholesteatoma, but the rest of the mastoid appeared to be eburnized and was left untouched. At the end of seven weeks this ear was dry. It was very gratifying to have the hearing in this ear increase from eighteen to one hundred and ten centimeters (acoumeter).

In this case the facial nerve was involved before the operation, and the slight facial paralysis which remains cannot be attributed to the operation. Both the facial paralysis and the labyrinth infection would have been avoided by an earlier operation.

In nearly all of the above cases radiographs were taken of both ears, and in the cases in which only one mastoid was involved the normal ear furnished a standard for comparison. Invariably the diseased mastoid gave a dimmer picture than the normal one, the mastoid cells were indistinct, and the cell walls were partly or completely obliterated; whereas in the normal ear the cells were clear cut and distinct.

Stereoscopic radiographs of the head, taken to show the infected mastoid, were found to be exceedingly valuable helps in making the diagnosis and in determining the position of the sigmoid sinus, the relation of the mastoid cells to it, and how far forward the cells extended into the zygoma, and also the lower limit of the brain cavity over the middle ear and the antrum.

It is possible in many cases to make a positive diagnosis of exposure of the dura and sinus by studying the stereoscopic radiographs of the mastoid before such exposure has caused any clinical symptoms. In other words, stereopscopic radiographs often enable us to recognize the imminent danger of meningitis, sinus thrombosis and brain abscess before these infections actually develop, and by prompt operation to prevent their development.

In the acute cases the operative findings and the results obtained justify the following conclusions: A profuse purulent discharge from the ear continuing for more than six weeks is almost positive evidence of mastoid infection. If, in addition to this, there is some sagging of the posterior canal wall, the evidence of mastoid infection is certain. The sooner a mastoid operation is done the better is the prognosis for rapid recovery with normal hearing.

Elevation of the temperature, if present, helps to confirm the diagnosis; but a normal temperature does not speak against the diagnosis of mastoiditis.

Tenderness over the mastoid antrum or tip or over the root of the zygoma and fixation of the sternocleidomastoid muscles are all confirmatory signs.

In chronic suppurative otitis media, careful, persistent local treatment should be carried out for two months, if the condition improves during this time. If it does not improve in two months' time, or if there are any positive indications of involvement of the labyrinth, sinus or brain, then a radical mastoid operation should be done.

It is hardly necessary to state that these conclusions are not original with the author. They are the accepted teachings of all otologists.

XXXIX.

RADIOGRAPHY AS AN AID IN THE DIAGNOSIS OF MASTOID DISEASE.*

By Frederick M. Law, M. D.,

New York.

The radiograph alone cannot be depended upon in the diagnosis of mastoiditis, as there are various conditions which influence the appearance of the plate; but used in conjunction with other methods of diagnosis, it is an invaluable aid.

In order to interpret a plate correctly, one must be familiar with the clinical symptoms; therefore, when a patient is sent to the radiographer, a short note of the clinical findings should accompany the patient.

The normal mastoid is a pneumatic cavity containing bony partitions, hence there is very little resistance to the passage of the rays; the silver of the plate is easily affected and a dark area is produced; the bony partitions showing as light lines. The lateral sinus and emissary vein, being really large cavities, show as dark streaks in the bony structure of the skull, but where the sinus crosses the normal mastoid cells, the additional cavity of the sinus is merged with the cells and the outline is distinguished with difficulty.

A mastoid structure that is filled with pathologic material or covered with a hard cortex offers considerable resistance to the passage of the rays; the silver of the plate is not acted upon and an area lighter than normal is produced. In such a condition the lateral sinus, being a cavity, allows more of the rays to pass and shows as a dark streak through the light area.

This appearance of the lateral sinus in the mastoid structure is often a diagnostic point in determining the difference between a normal mastoid and one filled with fine granulations.

The auditory canal, if free from pus, shows as a dark spot, but if pus is present, the outline is made out with difficulty; then the location must be determined from the articulation of the jaw, which is about one-quarter inch in front of the canal. The shadow of the pinna also aids in determining the position of the canal.

---

*Read before the New York Academy of Medicine, Section on Otology, May 12, 1913.

If the angle of the principal ray strikes the mastoid correctly, the entire mastoid region is shown clearly, and if the auditory canal happens to be straight and is perpendicular to the center of illumination, the small bones of the ear will stand out fairly sharp.

In the majority of cases the two mastoids are similar in structure; if there are large cells in one there will be large cells in the other. For this reason both mastoids are radiographed and the condition of the cells compared. The difference between the clear cells of the normal mastoid and the dull hazy cells of the diseased side is very plain.

The only conditions in which any doubt may arise is in the case of a double mastoid or an old chronic ear on both sides with an acute exacerbation in one. In these cases the cells of both sides are occluded and both plates show a hazy blur with the outline of the lateral sinus showing clearly. In such conditions dependence must be placed on the clinical symptoms, but if the plate is a good one, sclerosis may sometimes be determined by noting carefully the appearance of the shadow. The cells are completely destroyed and the mastoid resembles the bone of the skull; the skull and the mastoid seem to merge into one another and practically no outline of the mastoid can be seen, the anterior border of the lateral sinus standing out distinctly.

Mastoiditis is shown by the appearance of the cells as compared with the normal side. This may be from a slight hazy appearance to complete occlusion of the cells. In a mild condition there is a slight haze over the cells, but the bony partitions are still intact; as the condition progresses the haziness increases, light spots appear, which indicate collections of pus or spots of granulations. In a severe case the haziness becomes a dense white blur with no partitions visible, the lateral sinus standing out clear and the auditory canal barely showing. Cholesteatoma shows as a dark area in the midst of the white.

It is of course essential that the exposure of the two mastoids shall be similar and the density and contrast alike, or there will be difficulty in comparing the two sides.

It is difficult to locate accurately the position of the lateral sinus in a single plate made in the regular mastoid position, for the reason that some sinuses are placed deeper than others, and the deeper the sinus, the farther from the plate it is and the nearer the shadow is thrown to the auditory canal. Even so, the distortion will not be greater than one-eighth of an inch, and one can say the sinus is far forward or far back.

The location of the emissary vein is shown in the majority of cases.

A radiograph of the mastoid will show: The size and extent of the mastoid cells; the presence of pathologic material; sometimes the presence of sclerosis; the presence of cholesteatoma; the approximate size and position of the lateral sinus and emissary vein.

TECHNIC.

In making a radiograph the head is placed on a raised platform provided with a means for holding the head rigid; the ear to be radiographed is placed on the plate; the tube is centered so that the central ray will pass through the head to the mastoid on the opposite side on a line 15 degrees from the vertical and 15 degrees from the horizontal. This is an arbitrary rule which must be varied in some cases. With some patients the ear is placed higher than others, and in these the vertical angle can be lessened.

Some radiographers fold the pinna forward. If this is done the head is tilted slightly backward and the horizontal angle of the tube need not be so great.

A medium hard tube is used with sufficient exposure to produce good density and contrast.

XL.

A DISCUSSION OF THE MODUS OPERANDI OF GALVANIC NYSTAGMUS.*

By Lester Mead Hubby, M. D.,

New York.

Galvanism produces or affects nerve impulses through electrotonic currents or phases induced in the nerve. These cause phoretic changes in the tissues. Negative ions, such as oxygen, acids, etc., are drawn toward the anode; and positive ions, such as hydrogen, alkalies, etc., toward the cathode. Osmosis is altered, and may be reversed by this process during the passage of the current. Through the microscope a flow of fluid may be seen in a muscle fiber from the anode to the cathode, while the current is passing, with the production of a swelling at the cathode. ("Electricity," Chas. S. Potts.)

By these changes the nerve impulses, nerve irritability and conductivity are altered. In the anelectrotonic region the nerve irritability, conductivity, and strength of impulses are decreased; while in the catelectrotonic region the reverse is true.

Sufficient catelectrotonus of the normal vestibular nerve fibrils and terminations of one side of the head produces combined horizontal and rotatory nystagmus to that side of the head, whatever the position of the head. Sufficient anelectrotonus of the same results in nystagmus to the opposite side of the head.

Galvanism of both ears simultaneously with a double electrode causes no nystagmus unless one vestibular nerve, wholly or in part, is more sensitive than the other.

Catelectrotonus of one ear together with anelectrotonus of the other ear produces about double the amount of nystagmus, or results in nystagmus with about half the amount of amperage th[illegible] is required in galvanism of one ear alone.

In de[illegible] of the labyrinth of one side, the vestibular nerve, [illegible] functionating, generally requires greater

---

[illegible] York Academy of Medicine, April 11, 1913.

amperage to produce nystagmus than the nerve of the other side; and cathodal irritation of the normal side generally produces nystagmus with a slightly lower amperage than the anodal. This latter is explained by the presence of a more or less spontaneous nystagmus to that side.

The application of an electrode near the ear is at a great relative distance from the cristæ compared to the distances separating the latter, roughly speaking 5 cm. at least to less than 5 mm. respectively; and the diameter of the electrode generally used is greater than the greatest distance between the cristæ. Moreover, the galvanic current tends to diffuse in all directions from the point of application of the electrode.

Thus it is readily seen that all the vestibular nerve terminations are about equally affected in the ordinary method of testing.

Galvanic stimulation of individual cristæ or of the neighboring nerve fibrils is exceedingly difficult and in most cases impossible. It can only be done when the labyrinthine capsule is well exposed, as in a radical mastoid operation, and when the field can be made exceedingly dry, free from granulations, etc. I have succeeded in producing it in only one individual thus far. The cathode, an electrode about 1 mm. in diameter, was placed over the canals a few millimeters from their ampullæ seriatim. The different points of application were distant from each other about 5 to 6 mm.

As the cristæ are closer together than this, you can see how difficult it would be to sufficiently concentrate the current on one crista if applied near the cristæ themselves. By this method of application the current reaches a single crista, or at least the vestibular nerve branch going to the crista, in sufficiently concentrated power to produce a preponderating irritation of the nerve termination sought. (See chart No. 1.)

The galvanic current leaves a nerve in a condition of hyperirritability for a period lasting from a few seconds to fifteen minutes.

After the application of the anode the positive modification (Pflüger) lasts from a few seconds to several minutes.

There is a brief period of negative modification following cathodal irritation, followed by a positive modification lasting from one to fifteen minutes.

In positive modification the irritability of the nerve is increased, in negative modification, decreased.

This explains the fact that a doubtful spontaneous nystagmus is often increased to a demonstrable degree after galvanism of the ear.

In order to discuss the modus operandi of galvanic irritation of the vestibular nerves, it will now be necessary to consider the latter somewhat in a general way.

In the evolution and development of the vestibular nerve system, stimuli induced by head movements and changes in intralabyrinthine pressure were the only factors. The automatic interpretation of these impulses in the cerebellar and paracerebellar nuclei was always in terms of relative position and changing positions of the head.

Unnatural, unaccustomed stimuli, such as the application of cold or heat, or of the galvanic current, to a small area near the static labyrinth, inducing centripetal vestibular nerve impulses, could, therefore, only be interpreted as relative position and changing positions of the head. The same can be said of sufficient changes in intralabyrinthine pressure, however induced.

Conjugate deviation of the eyes (the slow component of nystagmus) and somatic muscular movements away from the canal stimulated and in the plane of that canal, are the automatic responses in all probability of the cerebellar cortex to such stimuli. For Bárány has demonstrated many of the reaction centers in this part of the cerebellum, and the slow component of the nystagmus is merely the reaction movement of the eyes. The rapid component of the nystagmus is the reflex central correction of the conjugate deviation of the eyes, and its centers are probably located in the posterior portions of the median frontal convolutions of the cerebral cortex, the location of the centers for voluntary movements of the eyes.

Nystagmus production is the result of the action of a long chain of neurons, and is not to be compared with the action of a single neuron, such as that of a motor nerve and its muscle. To remind you how long this chain is I have drawn a chart showing the probable connections, omitting the cerebral connections. You will see that at least twenty-three neurons are involved in the production of vestibular nystagmus, without counting the cerebral connections. (See chart No. 2.)

The combined centripetal impulses from the cristæ of both sides, under normal conditions of body rest, produce a certain condition or tonus of rest or equilibrium—a nystagmus equilibrium. If the impulses from one or more cristæ of one side are increased or decreased, relatively or absolutely, to a certain degree, this equilibrium is upset and nystagmus ensues until the equilibrium is reestablished.

This explains the long continued nystagmus following destruction of one static labyrinth.

In man the end organs of the vestibular nerves in the ampullæ of the semicircular canals are situated in the row of cells (sustentacular cells) with their hair-like prolongations into the hat-shaped gelatinous cupulæ. The hair cells are most numerous near the summits of the cristæ.

There are the following possible ways in which these nerve terminations can be irritated, namely—by variations in pressure, or stretching, or torsion, or combinations of any or all of these forces.

That variations in pressure must be a factor is evident from the following considerations:

1. Nystagmus occurs when there are sufficient changes in intralabyrinthine pressure, such as is produced in slight effusions of serum, pus or blood, in the static labyrinth, etc.

2. Ewald's experiment is not a simple production of endolymph movement, but with the latter there must be changes of endolymphatic pressure. For the membranous labyrinth is a closed tube, the endolymph is practically incompressible, the walls of the membranous labyrinth are surrounded by the practically incompressible perilymph, and the saccus endolymphaticus within the dura by the equally incompressible cerebrospinal fluid in the cranium.

Note.—Possibly the saccus endolymphaticus has the function of ameliorating great changes in pressure in the membranous labyrinth by its own expansion or shrinkage, acting thus as an equalizer of pressure.

Therefore. on forcing a piston into or against one membranous canal, the endolymphatic pressure must be raised, temporarily at least; likewise on suction the endolymphatic pressure must be lowered.

3. Some animal cristæ are apparently arranged so that they seem capable of responding only to changes in pressure. (Siredon Mexicanus, etc.)

It is not within the scope of this discussion to attempt to explain why pressure on the horizontal canal toward the ampulla has the reverse effect of that on the vertical canals. Suffice it to say that it is only necesasry to assume that the anatomic and histologic arrangement of the cristæ is such that movements of the endolymph on rotation of the head produce greater irritation in one direction than in the other, in the different canals.

Above normal irritation of any crista, whether induced by quick head movements, galvanism, rotation, disease, pressure, or caloric means, causes nystagmus to that side of the head.

On the other hand, if through any of the above agencies any crista is put in the condition of subnormal irritation, then nystagmus results to the opposite side of the head, not alone through overaction of the opposite side, but also through hypostimulation of the same side.

There must then be an average condition of rest, a nerve impulse tonus, if you will, of these nerve terminations in each crista, as suggested by Goltz for pressure only. For instance, each crista must be continuously transmitting impulses concerning the exact status of pressure, torsion, stretching, etc.

It is like a man wearing a hat—the weight of the hat and the compression of the hat band produce nerve impulses continuously to the brain. These impulses are soon disregarded. But if there is a sudden change, such as a gust of wind tugging at the hat, the nerve impulses are greatly changed, and more or less automatic reflexes result, such as grasping at the hat, etc.

So with the crista—its automatic responses are nystagmus and somatic muscular movements.

A certain amount of increase of nerve impulse tonus of any crista produces nystagmus toward that side, and somatic muscular movements away from the canal and in the plane of the canal; a certain amount of decrease of the nerve impulse tonus of a crista will produce nystagmus away from the canal affected, and somatic muscular movements toward and in the plane of the canal.

If we call irritants only those forces which increase the nerve impulse tonus of the cristæ of one side of the head, then we see that when they are exerted against one side of the head they cause conjugate movement of the eyes and somatic muscular movements away from the irritated side.

On the other hand, if depressants are applied to the tonus of the cristæ of one side of the head, the reverse occurs, namely, conjugate deviation of the eyes and somatic muscular movements toward the affected side.

Let us now consider why it is that in irritants such as disease, head movements, galvanism, etc., applied to all the canals of one side, the result is always a combined horizontal and rotatory nystagmus to that side.

If you will examine this model of the three canals, remembering that hyperirritation of one crista produces nystagmus toward that side of the head and in the plane of that canal, you will find the following:

Irritation of the right horizontal canal will produce horizontal nystagmus to that side; that of the superior canal will produce a rotary counter-clockwise movement of the eyes forward and to that side, which viewed from the front is rotatory to that side; irritation of the posterior canal will produce a rotary counter-clockwise nystagmus backward and to that side, which viewed from the front is rotatory to that side. Therefore, the resultant of these three must be a combined horizontal and rotatory nystagmus to the side irritated.

Bárány's explanation of galvanic nystagmus rests on the theory that there is a nystagmus equilibrium, and that there is altered conductivity of nerves in electrotonus. He claims that the slightest change in the nerve impulses from either side of the head upsets this equilibrium and results in nystagmus, the direction of the nystagmus being toward the side which is relatively stimulated. In catelectrotonus the conductivity of the vestibular nerve is increased. Therefore, nystagmus results toward that side on account of the increased transmission of the nerve impulse. In anelectrotonus the conductivity of the vestibular nerve is decreased. Therefore, the impulses are more or less blocked on that side, and those of the other side are relatively increased, and nystagmus results to the opposite side.

As pointed out by Brüning, if this theory were true, on the production of cold caloric nystagmus to the opposite side, the application of the cathode to the irritated side should increase the nystagmus to the opposite side, as the nerve conductivity is increased in catelectrotonus.

Instead of this, the nystagmus is stopped by the catelectrotonic effect on the nerve.

The same is true of heat caloric nystagmus to the same side being overcome in the production of anelectrotonus of that side.

Brüning's theory rests on the physical movement of matter in the neighborhood of the cristæ toward the cathode and away from the anode.

It would be difficult to harmonize this with the fact that nystagmus of the same kind and degree is produced, whether the electrotonus occurs near the cristæ or in the nerve itself when the cristæ have been destroyed.

With our present limited knowledge, the following theory seems to explain galvanic nystagmus in health and disease of the vestibular neurons:

1. Each crista with its hair-like processes is continuously transmitting afferent impulses of its condition in respect to pressure, stretching, or torsion, or combinations of these three. The condition of the nerve terminations in the crista, hair cells, etc., is then either in a normal state of irritation, a hyperirritated condition, or a hypoirritated condition.

2. The sum of these impulses from all the cristæ in body rest and under normal conditions, produces from the centers efferent impulses to the eyes, resulting in a nystagmus equilibrium.

3. Sufficient change in the relative amounts of these impulses upsets the nystagmus equilibrium and nystagmus results.

4. Impulses from any crista on one side of the head, however induced, indicating sufficient increase of irritation, cause nystagmus to that side of the head; those showing a sufficient decrease, cause nystagmus to the opposite side of the head.

5. The nerve impulses from the vestibular nerve and its terminations are altered during the production of electrotonus.

(a) Catelectrotonus induces a hyperirritated condition of the nerve and nerve terminations, an increase of afferent impulses; therefore, a nystagmus toward that side of the head.

(b) Anelectrotonus induces a hypoirritated condition of the nerve and nerve terminations, a decrease of afferent impulses; therefore, a nystagmus to the opposite side of the head.

6. The character of the nystagmus will vary with the relative amount of irritation of the different nerve terminations, cristæ, etc., and will be a resultant of the same. For instance, if the crista of the horizontal canal is mainly irritated, the nystagmus will approach the horizontal type.

Anterior View of Model.

Posterior View of Model.

XLI.

EXHIBITION OF RADIOGRAMS ILLUSTRATING THE PATHOLOGY OF MASTOIDITIS.†

By Henry S. Birkett, M. D.,

Montreal.

I wish to express to you my appreciation of the honor of the invitation extended to me to take part in this evening's discussion, but much regret that owing to a change from the original date to the present, it conflicts with the conduct of my examinations in the University, hence my reason for being absent.

When reflecting upon the value of radiography in diseases of the frontal sinuses and antra, it occurred to me that perhaps the same method might be applied to the mastoid process, and thus, independently of any work that had been done elsewhere, I had my first plate made in 1908. It was taken with the patient lying on his face, and the result was not satisfactory, as only the cells and the tip of the mastoid were visible. Studying the matter still further, I learned that Schier of Breslau had done some work along these lines, but his plates were taken with the patient lying on his side. In 1909 I again took up the work, having my plates taken with the patient lying on his side. For a description of the technic I am indebted to Dr. A. Howard Pirie in his article on "Radiography in the Diagnosis of Mastoid Disease,"* which is as follows:

"The patient should lie prone on a firm couch. The head is supported on an inclined plane making an angle of twenty-five degrees with the plane of the couch. The photographic plate rests on this inclined plane. The head is rotated 90 degrees, so that the patient looks directly to his side; this brings the mastoid into contact with the plate. The pinna of the ear is turned forward so as to obscure the mastoid as little as possible. The source of X-rays is placed vertically above the head, and the perpendicular ray is made to fall on a point two inches above the highest point of the pinna. The mastoid on each side must be skiagraphed separately. The glass of the focus-tube should be nine inches away from the hair. The exposure required will turn a Sabouraud pastille, placed at 2 centimeters from the glass, to one-third of the B tint. A medium hard focus-tube (4 to 5 Benoist) with 30 milliamperes for fifteen seconds from a Snook apparatus, gives a plate which should be fully developed in seven minutes (Ilford plate and developer). Skiagraphs of both right and left mastoids must be made of every case, as a single skiagraph of one mastoid is of little value. A different focus-tube should be used for each mastoid, as it is rarely possible to get one tube to remain constant in vacuum for both exposures. Both focus-tubes must, of course, be of the same hardness. This is one of the most important points in the technic, viz., to have two similar tubes of equal hardness and quality. The American made tubes lend themselves to this better than any others I have used."

---

†Presented before the New York Academy of Medicine, Section on Otology, May 12, 1913.

*Reprinted from "Archives of the Roentgen Rays," September, 1912.

The value of radiography in affections of the mastoid process is to demonstrate first of all the anatomic characters of the mastoid process itself, diploetic, pneumatic or sclerosed. It further gives us in many cases the position of the antrum and the position of the lateral sinus in its relation to the mastoid process, and in many, as shown in some of the plates, the emissary vein is distinctly seen.

Clinically, we are enabled by radiography to recognize when the mastoid process has become involved in acute inflammatory or purulent infection, by a distinct blurring or veiling of the cells. Should the disease have existed for some time, leading to destruction of the cells, this is often indicated by distinctly enlarged cavities, which occurring over the lateral sinus in some cases shows it definitely exposed as a dark area. One case I have on record—unfortunately the plate is not on exhibition owing to its having been broken—distinctly shows a sinus thrombus. This was evidently due to the fact that the thrombus was not only surrounded but infected with pus.

I wish, however, to make it distinctly clear that I am not an advocate of radiography as applied to diagnosing diseases of the mastoid process exclusive of one's clinical experience; I only look upon it as one of the nice adjuncts to one's work and I do not allow my judgment to be biased by any findings of the radiogram. But I will say this, that my readings of the radiograms have in operative cases always been confirmed.

I wish to acknowledge the valuable assistance of Mr. McNeil of the X-ray department of the Royal Victoria Hospital in the development of this work from its very beginning.

XLII.

FURTHER OBSERVATIONS ON THE TECHNIC IN THE AFTER-CARE OF THE RADICAL MASTOID OPERATION.

By Philip Hammond, M. D.,

Boston.

To achieve progress in any endeavor it is necessary to keep constantly in mind the goal toward which we are striving. Under no condition should we allow our efforts to be diverted from the main object by attempts, however zealous, to prove the truth of preconceived notions, unless those notions lead in the most direct way to the solution of the questions under consideration.

Pedantic as this may seem, it strikes directly at the cause of many exploited "methods"; for often one who has been instrumental in the development of an idea, or of some operative procedure, will become so fixed in his habits that he will tolerate no change in his accustomed routine, even though inconceivably superior methods have been adopted by his co-workers.

The advisability, nay even the necessity, of constantly changing one's mental attitude toward the treatment of the radically operated mastoid has been impressed upon the writer during an experience covering practically twenty years; if we but analyze the progress that has been made in this time, we shall see that it has come about by attempts to accomplish a definite result in the simplest possible way.

Even with the first cases it was recognized that success could not be attained without epidermization of the cavity, and to this end a postauricular opening was left through which various cutaneous flaps were introduced to aid the epidermis from the meatus in spreading throughout the inside of the bone.

Following the tendency of that time, the wounds were packed almost daily with gauze, and needless to say the granulations thrived upon this treatment. Then to aid in the process of epidermization, small Thiersch grafts or islands of epidermis were introduced. The permanent opening behind the ear proved so disfiguring that it was soon abandoned.

Opinions still vary as to certain details of the complete radical, which will be taken up later, but authorities are agreed up to a certain point. It is generally conceded that the bone operation should be most thoroughly and painstakingly performed, all crevices smoothed out, and all projections removed. Some form of plastic operation is then performed upon the posterior part of the cartilaginous meatus, to serve the double purpose of free access to the cavity for inspection and treatment and to furnish a dermal covering for a part at least of the mastoid. The type of this operation varies with the demands of the case and the habits of the operator, but the tendency is to obtain a meatus that will be sufficiently large to readily allow of all after-treatment of the entire cavity. The posterior wound is now generally sutured throughout, with the object of obtaining healing by first intention. The method of suturing that has given by far the best results in the hands of the writer is by means of the small metal clamp, or Michel suture. Not only is the apposition much better than that generally obtained, but they can be applied much more rapidly than ordinary sutures. Only one failure has been noted in an extended series, and that was in a case that had been subjected to repeated operations. With the object of preventing hemorrhage from the incision, a dilute solution of adrenalin chlorid was injected subcutaneously over the operative field as a preliminary, after the method of Jansen. There was a general breaking down of the wound and some sloughing; but this could not properly be attributed to the method of suture.

From this point on we encounter the greatest diversity of opinion as to the proper course to follow. Roughly speaking, the exponents of the various methods may be divided into two classes: those who use the skin graft to aid nature, and those who do not.

Before attempting to take any stand on the subject we should remember, first, that our whole aim is to favor the complete epidermization of the cavity, and secondly, that this should be accomplished as speedily and with as little distress to the patient as possible. The question then rapidly narrows down to one of results. Which method, or combination of methods, is most likely to show a favorable outcome? In this query we are likely to be led astray by prejudice, or by the fact that familiarity with a certain routine may have made the operator an expert with that particular method. Under these conditions any observer can scarcely avoid a bias toward the procedure which produces the best results at his hands, although the skill with which he administers his technic may be more of a factor in the successful issue than the means adopted.

To counteract this tendency, and to overcome as far as possible the objections which necessarily obtain where the operative work is carried on at the hands of one man, some of my colleagues at the Massachusetts Charitable Eye and Ear Infirmary have joined with me in an attempt to search out the truth of this matter. Cases have been operated upon by all the methods and the results noted. In addition to this, as many as possible of the cases operated upon for several years past have been requested to report at the hospital for observation. These cases have been carefully examined and the results freely discussed by advocates of the different methods of operation; so that the cases have been subjected to the criticism of the opponents as well as of the partisans of the methods employed. As a result of this inquiry several facts seem to have been established: first, the end results do not appear to favor any particular procedure, although the length of treatment was lessened in the cases subjected to grafting. Second, the cases in which the primary graft was used apparently did as well as those in which it was applied at a later stage. Third, as was to be expected, the best results were observed in cases where the most careful bone work had been done.

One of the interesting observations arising from a study of this series was the comparison of the very thorough exenteration practiced today with that done several years ago; the most recent cases allow of much better access to the middle ear, and the complete removal of the facial ridge leaves an extremely smooth cavity. This naturally aided in the healing of the wound, particularly where skin grafts were used. Many of the older cases had prominent ridges left at the site of the posterior canal wall, not only greatly interfering with the inspection of the ear, but actually handicapping the recovery.

Another point on which improvement has recently been made is in adopting some device to keep the meatus permanently enlarged. In a majority of the older cases it had narrowed down so that it did not provide room for thorough inspection of the cavity. In the hands of the writer the best results have been obtained by the use of a Koerner flap. This is carried well into the concha, the base of the flap being made as wide as possible. The resulting tongue is then pulled forward out of the meatus, and the skin of the flap separated from the cartilage, much as a submucous operation is done in the nose. The cartilage is now removed from the flap to its base. A deep buried chromicized gut suture is then passed through the base of the flap from behind and caught into the periosteum of the posterior edge of the wound, thus holding the meatus well open. To overcome the rather pointed corners in this artificial meatus, a short piece of large caliber rubber tubing is slit lengthwise, so that it is in cross-section like the letter C and can be expanded to fit any size canal. This is placed just at the entrance of the meatus and completely filled with gauze, continuous with the packing of the wound. It serves the double purpose of giving a better contour to the meatal edge, and of preventing the packing becoming so adherent to the edge of the wound.

While in the exceptional case very nearly if not quite as rapid healing takes place in the non skin-grafted cavity, this result is by no means uniform. With a reasonably small cavity, and flaps so placed from the canal as to advantageously assist in the spread of new epidermis, a favorable result is almost assured without further assistance. This is quite in accord with the teachings of general surgery; but one must not expect the skin to bridge a gap of more than half an inch.

With a large cavity it would seem to be only reasonable to expect that a graft might render valuable assistance in shortening the treatment. Indeed, the brilliant results which followed the use of some of the first Thiersch grafts for this purpose led many operators to adopt this method in all cases. Some always apply the graft at once, others as invariably await a later stage, commonly from the fourth to the tenth day. One operator advises further curettement of the cavity at this time, as a more certain means of encouraging the graft to take.

That some, at least, of this abundance of detail is needless, is attested by the results in certain cases, which from neglect have disappeared from observation almost immediately after operation, and without skin graft, only to reappear after months or years with a completely epidermatized cavity.

It is thought by many, however, that a suitable graft, successfully placed, lessens the period of convalescence decidedly. The chief objection advanced against its employment seems to rest on the fact that some operators experience a difficulty in having it "take."

Due care in the preparation of the cavity, however, will materially aid in accomplishing a successful result. If, under the best of conditions failures occur, something is wrong with the technic. In this the writer speaks not alone from his own experience, but from that of his colleagues as well. In the hands of several operators at our hospital the results are practically uniform.

No attempt is made at the present time to apply separate small grafts. A large thin graft, two inches or more square, is cut from the thigh with an ordinary amputating knife. The skin is immediately spread out on the place from which it was cut, exactly as if it had not been severed from its attachments. A square of crepe de lise is then laid on it, and the skin and crepe together removed from the thigh. This simple device absolutely prevents rolling or curling of the edges. A piece of ordinary gauze—or rather, several layers which have previously been rolled into a cone are then placed under one corner of the crepe and skin, in such a manner that the entire apex of the cone and at least one side is completely covered with skin, the raw side out. One side of the cone is purposely left free of skin, as this side goes into apposition with the anterior wall of the canal, which is already covered with epidermis. The cone, or plug, is then introduced through the meatus, the point guided toward the middle ear, and then the gauze of the plug gradually fed into the cavity, expanding and completely filling it.

It must be remembered that the graft is adherent to a thin, smooth, silky crepe, and the pressure of the mass of gauze causes this to reach every part of the cavity, even where the meatus is much smaller than the inside, the gauze expanding in much the way that a bristle probang does. This gauze is allowed to remain undisturbed three or four days, at the end of which it is gently removed. The crepe de lise comes with it, but the skin never. In the interval it has become firmly attached to all denuded surface. There are naturally some loose ends where the graft is superimposed upon the epidermis of the meatus, and there are necessarily reduplications or folds in the graft, as it is impossible to adapt a flat surface to the inside of a sphere without such. These loose pieces finally slough out, and during the process there is a very active suppuration to deal with. In most of the cases the superficial layers of graft are cast off, but underneath there is the firm pink appearance of new skin, rapidly paling out. Immediately following the removal of the gauze plug it is possible to demonstrate that the graft has "taken" by pulling upon it. It stands a very considerable strain, and if we succeed in detaching a piece, it causes bleeding, showing that it is definitely attached to its new resting place.

This method of applying the graft was described by the writer several years ago, and has been in use continuously since. It appears to be simpler in execution than the Ballance method, and quite as certain of a successful result. The graft may be applied either at the primary operation or at a later period.

Following the throwing off of the superficial layers of epithelium a simple cleansing of the cavity with hydrogen peroxid may be permitted, but aside from keeping out the products of suppuration, the less treatment given the better. The writer does not favor the use of drying powders, scarlet salve or other medications, as they are apt to produce crusting and deceive one as to the true condition of the cavity. Under no conditions should packing of the cavity be continued.

Cases where the skin does not "take" pretty generally throughout the cavity are rare; but if such occur, and granulations form, they should be kept down by the free use of caustics. This is substantially the treatment that must be followed if no skin graft be used.

In summing up the record of progress, emphasis should be laid on the fact that while once the radical mastoid operation was looked upon as too severe a procedure to which to submit our private patients, it is now so treated that recovery takes place in as short a space of time as in the acute mastoid: and that while once the dressings were painful, protracted, and called for the exercise of as much skill as the operation itself, now, thanks to our modified technic, they are practically painless, and every contingency has been so carefully guarded against in the operation that only a minimum amount of after-care is required.

REFERENCES.

Ballance: Transactions Ninth International Otological Congress, 1912.
Freeman: Skin Grafting, 1912.
Hammond: Journal American Medical Association, November 17 1906.

XLIII.

THE FAUCIAL TONSILS AS A FOCUS FOR SYSTEMIC INFECTION.*

By George E. Shambaugh, M. D.,

Chicago.

The close relation between acute rheumatic fever and sore throat has long been recognized. Formerly it was often assumed that the sore throat occurring in connection with acute rheumatism was but a local manifestation in the pharynx of the general systemic condition. It is now generally believed, however, that the sore throat in these cases represents the focus of entrance for the systemic infection. Furthermore, it is now quite generally recognized that sore throat, for the most part acute tonsillitis, is very often the immediate cause for other systemic infections such as acute endocarditis and acute nephritis. It has not been so generally appreciated that the faucial tonsils are very frequently the focus for chronic systemic infections such as chronic arthritis, commonly known as arthritis deformans, chronic neuritis, cardiovascular degeneration and chronic nephritis.

In the *Archives of Internal Medicine,* April, 1912, Dr. Frank Billings, of Chicago, discusses the subject of "Chronic Focal Infections and their Etiologic Relations to Arthritis and Nephritis." This work was done on patients in the Presbyterian Hospital of our city, and it has been my privilege to examine the throats of many of them and to enucleate the faucial tonsils in certain of the cases where these structures were suspected of harboring foci for the systemic infection. The observations on these cases, and on a large number of other cases of focal systemic infection referred to me during the past few years, have impressed on me certain facts. First, as regards the relation of the faucial tonsils to systemic infection; second, as regards the conditions found in the tonsils in cases where these structures were clearly shown to harbor foci for systemic infection, and third, as regards the proper treatment for tonsils suspected of containing foci producing systemic infection. In this paper I wish to discuss briefly some of the conclusions which I have reached regarding these questions.

---

*Read before the American Laryngological Association, Washington, D. C., May, 1913

In the first place, I believe that the general practitioner, as well as the specialist, has not, as a rule, fully appreciated the importance of the relation existing between infections, acute as well as latent, of the faucial tonsils and certain systemic conditions. I believe that in general the importance of this relation has been more fully appreciated by the leading internists than it has by the specialists. This is, perhaps, to be expected, because the consulting internist is the one to whom such cases are referred. The internist is, moreover, familiar with systemic conditions caused by other foci of infection, such as abscess of the gums, chronic appendicitis, infection of the genital tract, cholecystitis, etc. The internist is, therefore, prepared to recognize in a structure like the faucial tonsil, which is so frequently the seat of violent acute streptococcus infection, not only the source for acute systemic infection, such as acute rheumatic fever, acute endocarditis and nephritis, but also the seat of persistent latent chronic foci of infection capable of causing chronic arthritis, chronic neuritis, cardiovascular degenerations, nephritis, etc. The attention of the specialist, on the other hand, is naturally centered more on the purely local conditions. He cannot be expected to have that fuller appreciation of the systemic conditions which experience brings to the internist. It is so easy for the specialist to conclude that because he has not in his experience recognized systemic conditions resulting from tonsil infection, therefore these systemic conditions do not result from disease of the tonsils. No greater mistake in reasoning than this can be made. It would be just as logical for an otologist to assert that because he has never seen a brain abscess develop in any of his cases of purulent otitis media, therefore purulent otitis media is not the chief cause of brain abscess. Dr. Billings (l. c.) states: "There can be no other reason for the prevalence of rheumatism found in childhood than the frequency of local infections in the throat and nose. Quite as frequently children have endocarditis without other symptoms of rheumatic infection, which has its source in the throat. I think anyone who has noted the difference in the physical and mental make-up of a child before and after the removal of large amounts of lymphoid tissue in the throat, must be convinced that the change is due not only to a better air space, but also probably to the removal of a constant, even though it may be slight, systemic infection."

As regards the appearance of the faucial tonsil which is a focus for systemic infection, it is quite clear that the size of the tonsil is no index of the menace this structure may be to the individual. A large tonsil is often chronically diseased or the seat of repeated violent acute infections and, therefore, a constant menace as a focus for systemic infection. On the other hand, one sees a great many very large tonsils where the whole appearance is one of simple hypertrophy, and where there is no history of attacks of acute tonsillitis. The danger from tonsils of this type is, as a rule, very slight when compared with the danger from a small tonsil which is subject to repeated acute infections. At the same time the children with these enormous, apparently harmless, faucial tonsils often do appear illnourished and generally below par. Very frequently, too, in such cases the removal of the tonsils results in such an immediate and astonishing improvement in general health that it can hardly be accounted for except on the assumption that these enlarged, harmless appearing tonsils often contain foci for a mild systemic infection. The enlarged tonsil in the adult, which I have come to regard with suspicion as the possible focus for chronic systemic infection, is often more or less edematous and congested. The surface is more irregular, caused at times by deep ragged fissures, at other times by swelling of the parenchyma between the meshes of hypertrophic connective tissue stroma. In many of the tonsils there are dilated crypts containing the characteristic foul smelling, cheesy deposits which in many cases from time to time produce circumscribed acute infection, as shown by the local congestion and soreness. At such times the patient experiences more or less general malaise and dullness. Not infrequently one can, by pressing on the base of a tonsil, express from the depth a creamy fluid exudate which is largely pus, often filled with virulent streptococci. These enlarged tonsils in the adult are frequently quite fibrous and hard, but in some the swelling is largely of the parenchyma and the tonsils remain soft and compressible.

Another suspicious type of tonsil frequently met with in adults is the very small fibrous tonsil. This condition is found in individuals who, for many years, have been subject to a great many attacks of acute tonsillitis. I have several times removed tonsils which were so shrunken as to represent scarcely more than a thin band of fibrous scar tissue, from which the macroscopic evidence of parenchyma had been largely eliminated and where the structure was so firmly adherent to the underlying tissue that it could not be pulled out of its bed, as is done before dissecting the tonsil free from the pillars. The patient is often not aware that the recurring attacks of sore throat are in reality recurring attacks of acute infection in the faucial tonsils. It is only by seeing the patient during an attack of sore throat that the real character of the trouble can be diagnosed. The amount of local discomfort, as well as the general symptoms, fever, headache, etc., are, as a rule, not very severe as compared with attacks of acute tonsil infection occurring in an enlarged faucial tonsil. I have seen a number of cases of chronic arthritis and chronic neuritis occurring in patients who had for years suffered from this type of tonsil trouble, where each fresh attack of tonsillitis was followed in a few days by an acute exacerbation of the chronic systemic infection, and where the enucleation of the small insignificant looking fibrous tonsils resulted in a complete cessation of attacks of acute sore throat as well as a disappearance of the chronic systemic infection and a remarkable improvement in the patient's general health.

Everyone is familiar with the small tonsil embedded under a fold from the anterior pillar and the tonsil with a deep horizontal fissure separating the upper from the middle thirds, and with the fact that such tonsils are unusually susceptible to acute infections which, therefore, especially predispose them to the development of latent foci capable of causing systemic infection.

There is another type of faucial tonsil that I have learned to regard as a frequent source for systemic infection. This is the tonsil stub remaining after partial removal or where the tonsil has been subjected to ignipuncture or surface cauterization. In these cases the surface scar seems often to predispose to the retention of infection in the pockets of the remaining stub, causing repeated attacks of acute tonsillitis as well as systemic infection.

Occasionally one observes near the surface of the tonsils a sealed pocket filled with yellow pus. The tissue immediately surrounding such a pocket of pus often shows from time to time an acute reaction with swelling and congestion. On the other hand, such pockets are often observed when there is no evidence of an acute condition in the tonsils which would suggest tonsil infection. Pockets of this sort in the faucial tonsils frequently constitute the foci for chronic systemic infection. The size of the pocket of pus is not an important factor. Small foci seem to be as dangerous as a larger abscess. It is often overlooked that such foci may develop in the depth of a tonsil. As a matter of fact, one discovers such foci much more frequently near the bottom of the tonsil than near the surface. It would seem that the foci developing near the surface of the tonsil find an opening and heal more readily than when they develop near the bottom of the crypts. The presence of latent foci can sometimes be suspected from the appearanec of the tonsil as described above. On the other hand, I have often been surprised after removal of a tonsil suspected of causing systemic infection to discover near the base a pocket of pus, when neither the surface appearance of the tonsil nor the history suggested tonsil infection. Such experiences have taught me not to be too dogmatic in asserting that a tonsil may not be the focus for systemic infection simply because there is nothing suspicious in its appearance.

In this connection I wish to call attention to a condition which may occasionally follow what seems to be a complete enucleation of the tonsil. This is the formation, in the process of healing and contraction, of a small fistula which later may become the seat of an acute purulent infection with a circumscribed abscess formation in the bottom of the fistula. This condition I have seen in three cases. In two of the cases the abscess was located about the middle of the tonsil region, and the acute reaction occurred more than a year after the tonsil enucleation. In the third case the fistula was observed three months after the tonsil operation. There had been no acute symptoms, and the fistula led to a small cyst the size of a pea, filled with pus containing staphylococcus, and located in the region of the supratonsillar fossa.[1]

Chronic foci of infection in the faucial tonsil, just as such foci occurring elsewhere, represent a condition which has had its origin in some acute infection. In most of the cases the patient gives a history of such acute infections, consisting in attacks of acute tonsillitis. In many of the cases with chronic foci in the tonsils there may not have been an acute attack of tonsillitis for a number of years. In all cases of chronic systemic focal infection, where the competent internist is unable to discover evidence of foci elsewhere, the faucial tonsil should be suspected. Usually when the tonsils are at fault there will be a history of attacks of sore throat, tonsillitis, and a careful examination of the tonsils will disclose evidences of chronic infection.

As regards the proper treatment of faucial tonsils suspected of harboring foci giving rise to systemic infection, this should be the same as the treatment of such foci of infection occurring elsewhere in the body. The treatment consists in the thorough removal of the suspected foci. In the case of the faucial tonsils, I know of but one way by which this result can be assured. This consists in the thorough enucleation of the faucial tonsils. Whatever theoretical arguments may be urged against tonsillectomy as a routine procedure, when operating on enlarged faucial tonsils in children, such arguments should not prevent us from the thorough enucleation of faucial tonsils, whether they be large or small, when these structures are suspected of harboring foci causing systemic infection. Only in those cases, especially in adults, where the danger of a serious bleeding is increased because of a high blood pressure or a slow coagulation time, should the enucleation of the tonsil give place for less radical measures. Here we must be content with efforts to cleanse the tonsil pockets and possibly also with cautiously slitting them open. Surface cauterization or ignipuncture should never be resorted to, as these only tend to increase the trouble.

The enucleation of the faucial tonsils in children requires a general anesthesia. In adults I prefer, as a rule, to do the operation under local anesthesia, for the following reasons: The operation can be done with the patient in the upright position; the annoyance from bleeding is very much less; the operation can be done with less traumatism to the surrounding structures, and the discomfort from soreness is less than where general anesthesia is employed. All cases are operated at the hospital. The patient remains at the hospital over night, or for several days if he wishes. I begin usually about 2 p. m. When local anesthesia is to be used the patient goes without lunch in order to decrease the discomfort from gagging while working in the throat. A hypodermic containing one-sixth grain of morphin with atropin is given just before the patient is brought up to the operating room. I use a 5 per cent solution of cocain in 1/1000 adrenalin, rubbed over the attachment of the tonsil. A one-half per cent solution of novocain, to which a few drops of adrenalin are added, is freely injected under the mucous membrane along the margin of attachment on the tonsil on both the anterior and posterior pillars. A deep injection of this solution is made in the lymphoid tissue at the lower part of the tonsil, and another is made in the base of the tonsil directly through the anterior surface of the soft palate. This part of the work is done deliberately, and both tonsils are prepared for operation before an incision is made. I have used this method for over three years and have never seen any symptoms of poisoning such as an injection of even a small amount of cocain usually produces. Both tonsils are dissected free from the pillars before either is removed with the snare. In most cases the only part of the operation that is at all painful is when the snare is being used. For this reason it is important that the difficult part of the operation, the freeing of the tonsils from the pillars, should be done before either tonsil is enucleated. After a patient has once been hurt by the removal of one tonsil, he may be too nervous to allow the careful freeing of the second tonsil from the pillars. The separation of the tonsils from the pillars is accomplished with the least discomfort to the patient by using a straight scalpel, very sharp and with a rounded end. I find that in separating the left tonsil from the posterior pillar and the right from the anterior pillar, it is an advanatge to hold the scalpel in the left hand while the tonsil forceps are held in the right hand. The patient holds the tongue depressor. The snare to be used when removing the tonsils under local anesthesia should be one that can be closed instantly if desired. The slow closure of the snare is often too painful when local anesthesia is employed.

XLIV.

REPORT OF TWO CASES OF CEREBROSPINAL MENINGITIS PRESENTING NO CENTRAL NERVOUS PHENOMENA UNTIL SHORTLY BEFORE DEATH.

By T. Passmore Berens, M. D.,

New York.

Mr. X., age fifty-nine years, consulted me January 10, 1913. He complained of deafness in both ears and of pain in the right ear for a week, following influenza, from which he was convalescing. Examination of the ears revealed impacted cerumen, the removal of which restored his hearing to such an extent that he heard a whisper at three feet in the right ear and at eight feet in the left ear. The right membrana tympani was slightly reddened. He complained of headache, also of pain in the region of the right mastoid. There was no bulging of the membrana tympani, which was slightly injected, no edema, and no pain on deep pressure. The patient had two uncomfortable nights, then returned because of pain on January 12th. The temperature was 97.5°, the redness of the membrana tympani persisted. There was no bulging, no tenderness, but slight mucopurulent discharge from the accessory sinuses of the nose. His general appearance indicated a grave illness, and he was placed in bed in the Manhattan Eye, Ear and Throat Hospital, where myringotomy was performed under nitrous oxid anesthesia. The incision was not followed by discharge of pus. Nevertheless, he was irrigated with bichlorid 1/10,000; and in the course of six hours a profuse discharge of pus appeared in the canal. The infective organism was the staphylococcus. For the two following days the temperature was 99° and the discharge very profuse. On the third day tenderness developed over the tip and antrum of the mastoid, with slight drooping of the superior wall of the external auditory canal. Headache persisted and the patient seemed unduly apprehensive.

Under gas ether anesthesia a simple mastoid operation was performed January 18th. The mastoid process, which was extremely dense, with few cells, contained much pus. Softened bone, which was adherent to the knee of the lateral sinus, was removed. The tegmen of the antrum was softened and adherent to the dura. The wound was packed with iodoform gauze, and the patient was returned to bed in good condition.

The case progressed satisfactorily for two weeks, when there was an unexplained rise of temperature to 102°. There was no chill, no sweating. The wound was clean and healing nicely. That night he complained of marked pain in the muscles of the calf of the right leg. There was no redness, but there was tenderness. The next day pain developed in the muscles of the left calf; and there was considerable pain in the sciatic notch on this side, with backache. The tongue was deeply furred. There was marked indicanuria. The temperature was 99°. Free purgation and aspirin controlled the pains to such an extent that the patient went to the toilet and walked about the hospital. On February 4th (the seventeenth day after operation) the temperature rose to 101°, and there was an exacerbation of his pains. He complained again of headache, and that it hurt his eyes to read. Dr. Frank Van Fleet pronounced the eye grounds to be normal. The temperature then became irregular, ranging from normal to 102°. The pain left the legs and centered in the sciatic notch and in the lumbar region. There was no Kernig, no Babinsky. The patellar reflex was normal. There was no stiffness in the neck. Dr. A. B. Duel was called in consultation, and confirmed my findings, viz., a clean, healing wound, with nothing but the fever pointing to involvement of the central nervous system. Dr. Chas. H. Richardson confirmed my diagnosis of migrating muscular rheumatism. Free purgation and large doses of aspirin again gave considerable relief. The patient insisted on visiting the toilet, and seemed quite able to do so. The temperature continued to be irregular, ranging from 99° to 102°. The pulse varied with the temperature from 72 to 84; respiration 18 to 20.

On February 12th (the twenty-fifth day after operation) the patient was comfortable, and much less apprehensive than at any other time during his illness. The temperature was 100°, and he presented every evidence of a beginning convalescence.

In the evening of this day he went unaided to the toilet, which was adjoining his room; and told me that he felt better than he had felt for two weeks. At about midnight his temperature rose to 102°; pulse was 108, respirations 104, pupils irregular. He gradually sank into unconsciousness, until at 5 a. m. (on the twenty-sixth day after operation) he was comatose. I opened the original mastoid wound. It was nearly healed. The lateral sinus seemed healthy. The dura was exposed and incised. The wound through it filled immediately with a great hernia of the brain. Careful search with a brain knife failed to reveal pus in the brain. A lumbar puncture brought forth a large amount of creamy fluid under great pressure. The patient died in a few hours, without having regained consciousness.

Autopsy was not allowed, but extensive examination through the wound failed to reveal an abscess. Much creamy fluid was recovered from the ventricles.

During the course of the illness there were frequent blood counts and frequent blood cultures—all negative. Widal and Wassermann were negative.

Examination of the cerebrospinal fluid resulted in the following report:

"Appearance, mucopurulent, with considerable admixture of blood.

Copper reducing substance, absent.

Lactic acid test, strong reaction.

Cytologic Examination.—About 10 per cent bulk of pus cells on centrifuging. These cells were largely polymorphonuclear leucocytes, with a few lymphocytes and endothelial cells.

Bacteria.—In the stained smear were many chains of streptococcus mucosus capsulatus, with an unusually heavy capsule, so heavy as to leave their identity in doubt until stained by Gram's method. These grew freely on blood serum.

Stain for tubercle bacilli, negative.

(Signed) J. G. Callison, M. D.,
Pathologist."

G. T., male, age nineteen years, was admitted to my service in the Manhattan Eye, Ear and Throat Hospital December 5, 1912, with the following history: When an infant he fell, striking the back of the head. He was a delicate child, and did not develop well physically or mentally. The lack of mental development continued and he became an idiot. During early childhood he had measles, with a discharging ear which supposedly healed. Scarlet fever developed a few years later, accompanied by a discharging right ear. The discharge continued until the time of admission. No more complete history could be obtained because of the extreme ignorance of the patient's relatives.

Examination of the right ear revealed a large mass of spongy granulations nearly filling the canal; mastoid tender; deafness; no vertigo; no nystagmus. The temperature was normal.

A Schwartze-Stacke mastoid operation was performed under gas ether anesthesia by Dr. S. McCullagh. The external auditory canal, middle ear and attic were found to be filled with granulations, and the ossicles were absent. The position of the dura was unusually low, and the lateral sinus was far forward. The antrum was absent. There was a large exposure of the dura and of the lateral sinus. The wound was packed and the patient was returned to bed. The next day the temperature rose to 102°, the patient became restless and complained of pain in the ear and in the back of the neck. There was no suggestion of central nervous involvement. The general condition was good. The temperature dropped to normal on the third day, and for three days following pursued an irregular course, ranging from 99° to 101°. On the fifth day the temperature rose to 103°. The wound looked healthy and clean. The patient remained restless and persisted in rising from his bed. No rigidities were apparent. Babinsky and Kernig symptoms were absent. The eye grounds were normal. The blood culture was negative. Blood count: Leucocytes, 18,100; large mononuclear leucocytes, 5 per cent; small mononuclear leucocytes, 9 per cent; polynuclear neutrophiles, 86 per cent.

For the three weeks following, the temperature ranged irregularly from 100° to 102°. Other symptoms, though sought for, were not found. The eye grounds remained normal. The restlessness continued and the boy persisted in leaving the bed and in walking about. Typhoid and malaria fevers were eliminated by blood examinations. Many of my colleagues in the hospital saw the case in consultation. Dr. Hutton, the attending physician, made this note on the 20th of December: "Deep-seated suppuration; if intracranial, so located as to give no definite localizing signs. Heart, lungs and abdomen negative."

On December 28th the boy was found wandering in the hall of the hospital. On the following day there developed a fleeting external strabismus, and on the operated side slight facial paralysis. Ankle klonus and exaggerated knee jerk demonstrable. and Kernig's sign positive. Neck slightly resistant. The eye grounds were normal. A lumber puncture revealed the cerebrospinal fluid, which was under pressure, to be almost creamy in color and consistency.

The evidences of central nervous involvement did not increase. A few hours before he died he arose from his bed and appropriated articles belonging to other patients. He sank into coma an hour or two before death.

Autopsy revealed an unusually well developed cerebrospinal leptomeningitis.

Examination of the cerebrospinal fluid resulted in the following report:

"About 15 cc. recovered. 10 cc. sent to laboratory.

Appearance, greenish, creamy, mucopurulent.

Amount of sediment, 2½ cc. in 10 cc.

Copper reducing substance, absent.

Lactic acid test, very marked reaction.

Cytologic examination—Cellular elements are mostly polymorphonuclear leucocytes, with some lymphocytes and endothelial cells.

Bacteria.—Large numbers of either streptococci or pneumococci, with some bacilli resembling B. pyocyaneus. These cocci were culturally proved to be streptococcus mucosus.

Stain for tubercle bacilli negative.

(Signed) J. G. Callison, M. D.,
Pathologist."

The postmortem examination, conducted by Dr. Kopetzky, revealed the spinal cord covered with a thick, creamy exudate, which extended into the ventricles of the brain. There was no involvement of the lateral sinus. The dura was normal over the seat of the mastoid operation. The internal auditory meatus was filled with granulation tissue and pus.

I have reported these two cases not because cerebrospinal leptomeningitis of otitic origin is a rare disease, but because in my experience, covering twenty-five years of otologic practice, I have not seen a single other case which did not present long before death some symptom other than fever pointing to involvement of the central nervous system. I confess myself at a complete loss to explain the lack of symptoms here presented. Brieger, quoted by Preysing, in the Transactions of the German Otological Association, 1912, says:

"In many cases of suppurative meningitis we see all the symptoms disappear after opening the labyrinth and after repeated lumbar puncture. I have seen such cases, which appeared almost in the last stages, seemingly completely recover. They got up out of bed, walked about, felt perfectly well; and we were astounded to see that within a few hours after a conditions of apparent complete well-being death should supervene. Autopsy revealed that in some cases there occurred in parts of the subarachnoid space, inaccessible to lumbar puncture or to other exploratory methods, a widespread plastic infiltration, while in other cases there existed, in addition to these localized infiltrations, diffuse inflammation of the meninges."

XLV.

CHOICE OF TREATMENT IN CHRONIC SUPPURATIVE OTITIS MEDIA.*

By George L. Richards, M. D.,

Fall River.

What is intended to be discussed is not the details of any operative or other treatment, but rather to what conclusion we should come with reference to the choice of treatment in cases of chronic purulent otitis.

First, in children. The records of all the ear clinics where children are treated show that many of them seek treatment for chronic discharging ears. On a history being sought, none will be obtained at first, but careful, skillful questioning will bring out the fact that the ear discharge has been the result of a cold, or has followed one of the exanthemata, measles or scarlet fever. If stated to be due to frequent colds, the first thing to ascertain is the presence or absence of adenoids or enlarged tonsils, and then to correct any abnormal pharynx or nasopharynx condition which may be evident or causative. Quite a number of cases clear up promptly with cessation of the discharge. Those which do not, and especially those due to the exanthemata, usually show on examination a considerable loss of the drum membrane and adhesion of the ossicles to the inner tympanic wall. Such cases should be treated for a while conservatively and carefully by such thorough cleansing methods as appeal to the individual treating the case. Unfortunately, this cleansing can only be done by the physician himself or by a nurse, and almost never by the patient or friend. This necessitates frequent visits to the physician's office or clinic, and is one of the drawbacks to the conservative treatment of chronic ears. Nevertheless, a large number of these cases will clear up, the discharge stop, and the hearing improve. Nature left to herself, if she has any reasonable opportunity for drainage, will usually wall off the diseased area from the sound area, and in the case of bone, either casts off the disease or forms a sclerosed wall between the diseased tissue and the sound area. While it is no doubt true, as William McEwen wrote many years ago, "that all cases of suppurative ear trouble are potential volcanoes," the fact remains that only relatively few of the actual number of cases either come to radical operation or die as a result of the disease.

---

*Presented before the American Laryngological, Rhinological and Otological Society, at its meeting held in Washington, May, 1913.

What next is to become of these cases of suppurative ear trouble which, after long and careful cleansing treatment, do not subside and do not clear up? What is to be recommended? Some type of operation, and, if so, what? In the choice of this, I think due consideration should be given to the question of how much hearing the child has—and many times the hearing is very good—and also to his or her ability to continue treatment. If the hearing is nearly or completely gone, the socalled radical operation is the operation of preference, as it comes nearest to complete surgical removal of the diseased material. Even here a more or less long course of after-treatment may be necessary, and it cannot be stated with absolute certainty that such an operation will always be successful in completely stopping the discharge. Many a case subjected to radical operation has occasional times when it suppurates, but as the drainage is good there is little danger from this, and if cases operated radically are seen occasionally there is little trouble.

When there is a fair degree of hearing and the major portion of the drums remains, I think some type of conservative surgical operation should be adopted, providing cholesteatoma is not present. By a conservative type I mean those advocated by Heath, Bondy, and Stacke, in which the drum membrane and ossicles are allowed to remain, and the tympanic cavity is drained from behind through the external canal. It seems to me that in America we have not yet sufficiently tried out this type of operation. I am aware that the socalled Heath operation has not fulfilled its expectations in the hands of others than its originator, but that is no reason for not continuing the search for a satisfactory conservative operation. While it is no doubt true that in some cases after a complete radical operation the hearing is not materially damaged, or may even be improved, yet reasoning from an anatomic standpoint it seems impossible that the hearing should be otherwise than usually made worse, and this I think will always be found to be the case if the affected ear is tested sufficiently carefully both before and after the operation.

How long ought a child's ear to be treated conservatively before some type of radical operation is performed? This is one of the hard questions before us. In our clinics patients with chronic ear discharge are asked day after day to return for what to the patient or parents or others seems a continual wiping out, without much result, and in the case of patients who come to our office and pay our fees, it seems to many of them a long drawn-out, expensive procedure, with, in many cases, nothing or little in the way of result. And when finally we suggest to them that after all an operation is required, they not infrequently and very naturally ask, "Well, if that is the case, why was it not done to begin with?" So that before beginning any conservative method of treatment an explanation, sufficiently simple to be understood by the patient, should be given him so as to forefend oneself.

The question will very naturally be asked, "If the ear becomes dry, will it stay dry and will I hear?" This, it seems to me, is largely a question of whether moisture, that is, water, can be kept out of the ear. After an experience of some twenty years in treating these cases, I am firmly of the conclusion that the drier we can keep them, the better and more rapid the results. You all know how difficult it is to satisfactorily dry an ear when you have the patient before you under most favorable conditions. When syringing the ear is ordered for home use, the ear is never dry, and it is practically impossible for anyone to properly dry the ear except under good illumination. What then is the result? A moist cavity at temperature of the body, and the microorganisms already present are only favored in their development by the conditions provided.

Second, in adults. When we turn to adults, we are confronted with two classes of chronic suppurative middle ear: one with, and one without cholesteatoma. Those without cholesteatoma may be handled somewhat the same as I suggested in children. An endeavor, first of all, is to be made to obtain a cure by conservative means, which with many things at our command for local use, and the careful cleansing and mild curetting which it is possible and permissible to do under good illumination, will, in most cases, be followed by good results. Here again the caution with reference to water and bathing is necessary. The question of hearing is not usually of great importance, unless both ears are involved, since if the adult has one good ear, and seeks treatment for discharge for the other, which is practically deaf, he does not usually expect the restoration of hearing in the affected ear, which is apt to be poor, and from which the discharge has usually continued for months or years before relief is sought. A radical operation is then to be advised in many cases.

The question of conservative treatment in the cholesteatoma case depends entirely on whether the opening is sufficiently large to allow the discharge of the cholesteatomatous mass. Where this is the case, and where the ear can be kept dry, even a cholesteatoma not infrequently can be successfully treated by conservative means. Where the opening is small and the cholesteatoma is moist, nothing short of a radical operation is to be advised. In the noncholesteatoma cases, most of them can be treated conservatively with results which are fairly good. In adults, as in the case of children, if conservatism is not followed after a reasonable time with good results, then the question of a radical operation is to be considered. It is surprising, however, what a large number of cases can be cured by careful attention to conservative methods. While I have no exact statistics, I am of the opinion that most of the cases of severe complications have occurred in patients where the suppurative otitic trouble has not been treated, but where it has been allowed to go on without much attention on the part of the individual until infection, through some channel or other, has reached either the brain, mastoid or lateral sinuses.

I have been interested to look over the statistics reported from some of the large ear hospitals with reference to the proportionate number of cases of purulent ear trouble which seek relief, as compared with the number of cases of operations done in these hospitals. The New York Eye and Ear Infirmary reports for the year 1911, 1283 cases of chronic suppurative otitis media, with 110 cases operated upon, a proportion of 8.5 per cent. The Manhattan Eye and Ear Infirmary reports of 1906-07-08-09-10 gives about the same results, 7350 cases, of which 706 were operated upon, or 10.4 per cent. The Massachusetts Eye and Ear Infirmary reports, so far as radical operation are concerned, for the years 1909-10-11, 6507 cases of chronic suppuration treated in the out-patient department, and with 80 radical operations, or 1.25 per cent. So that at least not over one in ten of those who seek relief, even at our large hospitals, are operated upon, and it seems to me that if thorough conservatism is applied, that in one's private practice more than nine out of ten can be cured without operation. Certainly the pendulum at the present moment seems to have swung back from the time when operation was suggested for nearly every case of chronic suppurative otitis to a somewhat middle ground, where cases manifestly demanding it are operated upon, while many others are cured, or at least the process brought to a standstill, without operation. In the final analysis each case must be judged on its own merits, due consideration being given to all the factors in evidence.

XLVI.

RADIOGRAPHIC FINDINGS ILLUSTRATING THE ANATOMIC DEVELOPMENT OF THE MASTOID BONE.*

(STEREOPTICON DEMONSTRATION.)

By William H. Stewart, M. D.

New York.

On December 29, 1910, I demonstrated before the Section on Laryngology and Rhinology a series of radiographs showing the development of the frontal sinuses. The cardinal points of the paper were the very early evidence of the frontal sinuses and the wide variations in size, location and existence. At the suggestion of your very able chairman, I have attempted to take up this line of work with the mastoids. I was stimulated by the general belief that the mastoid cells, under ten years of age, were represented by a spongy cancellous layer of bone, lying between the inner and outer table of the skull, called diploe.

Dr. Sidney Lange, one of the earliest workers on radiographic examination of the mastoid, says in his original article, read before the American Roentgen Ray Society on September 23, 1909: "Upon the mastoid skiagrams of children under ten years of age the mastoid appears entirely spongy and diploetic, there are no visible cells and the tip is undeveloped. The tip at this time consists simply of an inner and outer table with little cancellous bone between and shows no structure upon the skiagram. From ten to fifteen years of age the mastoid takes on the pneumatic characteristics, larger cells appear and grow downward toward the tip, its diploetic structure finally giving way to the more or less pneumatic adult type."

---

*Presented before the New York Academy of Medicine, Section on Otology, May 12, 1913.

Gray says: "The mastoid cells, like the other sinuses of the cranium, are not developed until after puberty."

Piersol says: "The mastoid process becomes fairly distinct in the course of the second year; it develops greatly about the time of puberty, when it becomes pneumatic. This may occur much earlier. J. J. Clark has seen it wholly pneumatic several times before the tenth year; once at three and one-half years of age."

The results of my investigations have been partially successful in demonstrating:

1. That we frequently have distinct and well formed pneumatic cells as early as two years.

2. That there is a wide variance in the character, location and extent of the mastoid cells under ten years.

3. That given six children of any age, say five years, no two will have mastoids alike.

4. That the generally accepted view that the cells are of the same character on both sides is not true in all cases—my experience with the normal will show that while the majority have a great similarity on both sides, that in about 15 per cent there is considerable variance in the character.

5. That the mastoid cells of the pneumatic type may extend into the occipital bone back of the sigmoid portion of the lateral sinus, into the squamous portion of the temporal, and above the external auditory canal to the root of the zygoma.

7. That with a fairly intelligent child the radiographic examination will clearly show the character and location of the mastoid cells in their relation to the lateral sinus, the external auditory canal and the tegmen tympani.

It may be interesting to quote the following observations in adults:

In Zuckerkandl's investigation of 250 temporal bones, the mastoid was found entirely pneumatic in 36.8 per cent. and wholly diploetic in 20 per cent. The remaining 43.2 per cent were mixed, the diploe being at the point of the mastoid and the cells above; neither size nor shape indicates its internal structure. This is somewhat in variance with Piersol, who quotes Randall in the study of 1000 temporal bones, in which he found scarcely 2 per cent of mastoid cells could be classed as diploetic, and only some 10 per cent as combining a notable amount of diploe with pneumatic spaces.

TECHNIC.

These radiographs were all taken of normal children from the age of one month to fifteen years, and I beg your indulgence if they seem comparatively poor, as it is no easy task to control a healthy child sufficiently to properly examine the mastoids with the X-ray. Extreme patience and indulgence are necessary. Success can only be achieved when you have the confidence of the child. This may be acquired in many ways. The method I pursued was as follows:

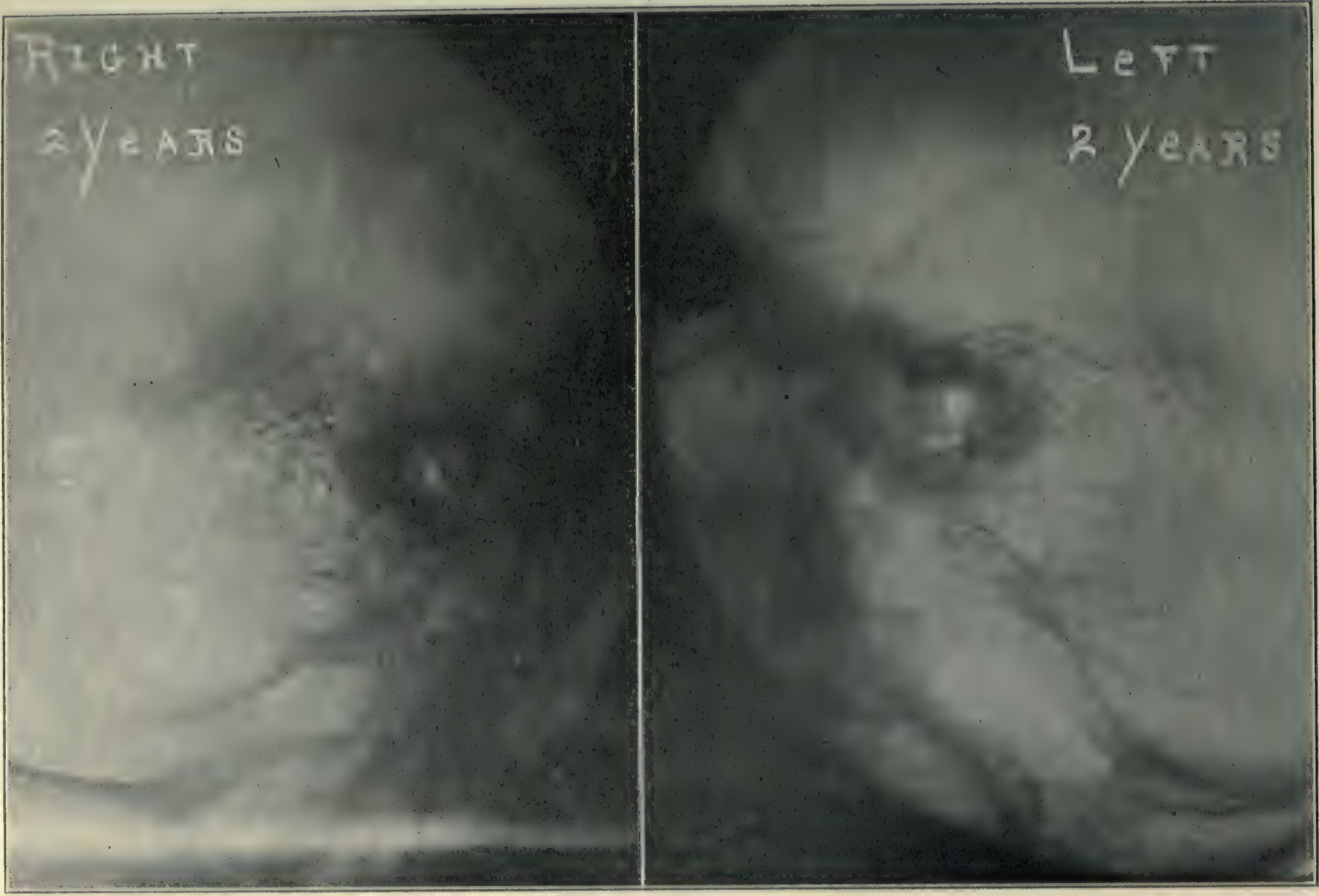
After repeated failure in not being able to obtain absolute immobility, I conceived the idea of having afternoon tea parties; one of the pleasures of this gathering was to have their "picture took." I have strenuously fought being relegated to a photographer; in this case I was obliged to be just an ordinary picture man. The children were gathered around so that they could all see the camera "shoot," that it did not hurt and that you must hold your head still.

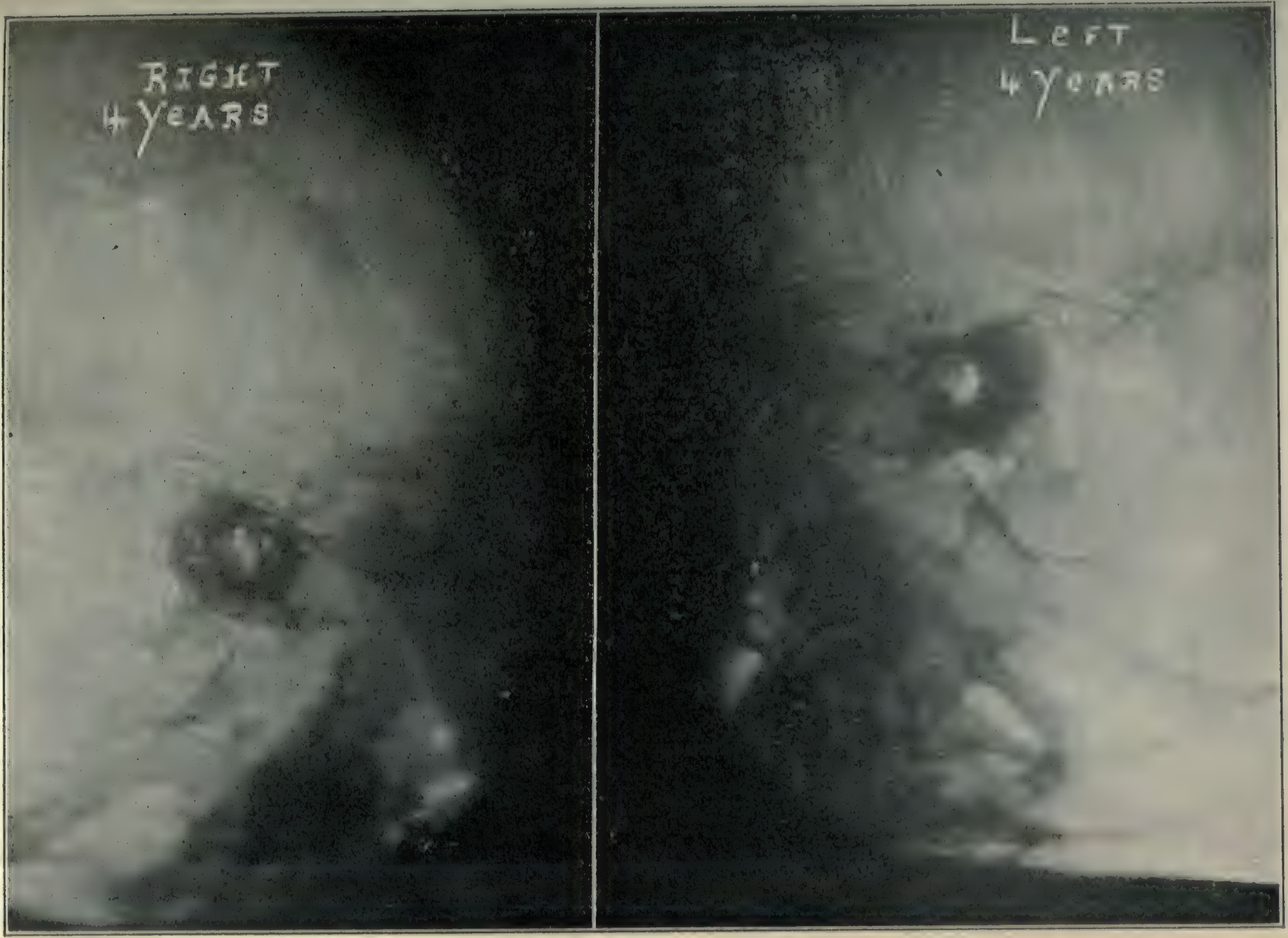
The child was laid on the table on its side, the head resting on the plate, placed on a wooden inclined plane of 25 degrees, the top of the plane at the neck; this brought the plate in direct contact with the mastoid region, the ear being pushed forward; a line drawn from the glabella through the middle of the tip of the nose to the symphysis of the chin was parallel to the upper surface of the table, which was supposed to be level. An X-ray tube of medium penetration with a vacuum that would take about 55 milliamperes was then adjusted in the tube stand, having a cone with an outlet of about four inches diameter attached; the axis of the cone being continuous with the central ray of the tube.

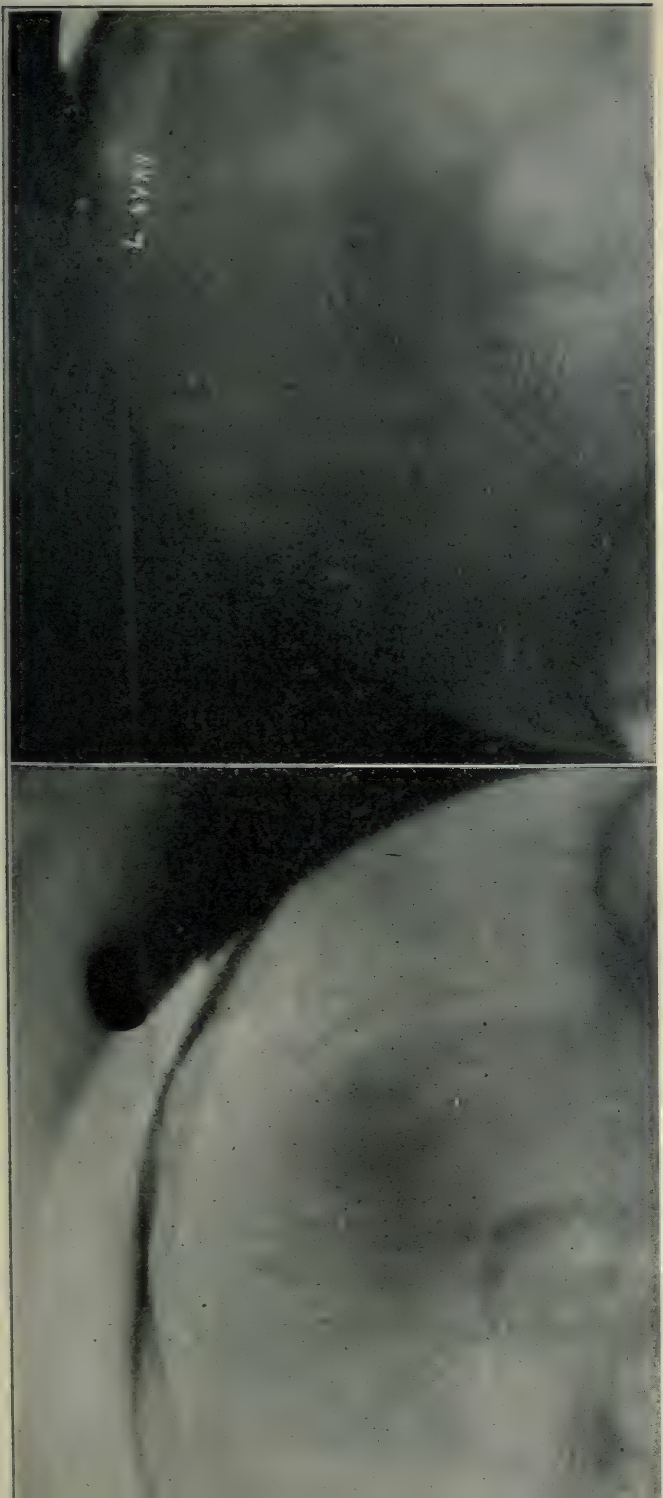
The cone, set so that the ray would be directed from above 10 degrees and behind about 15 degrees, was then moved over the head so that the axis would be centered at the point just below the parietal eminence on the upper side. An exposure is then made of about two seconds. This will, in the majority of cases, give us good radiographs of the mastoid with a minimum amount of distortion; it may be necessary to vary the angle somewhat, as we meet with different shaped heads.

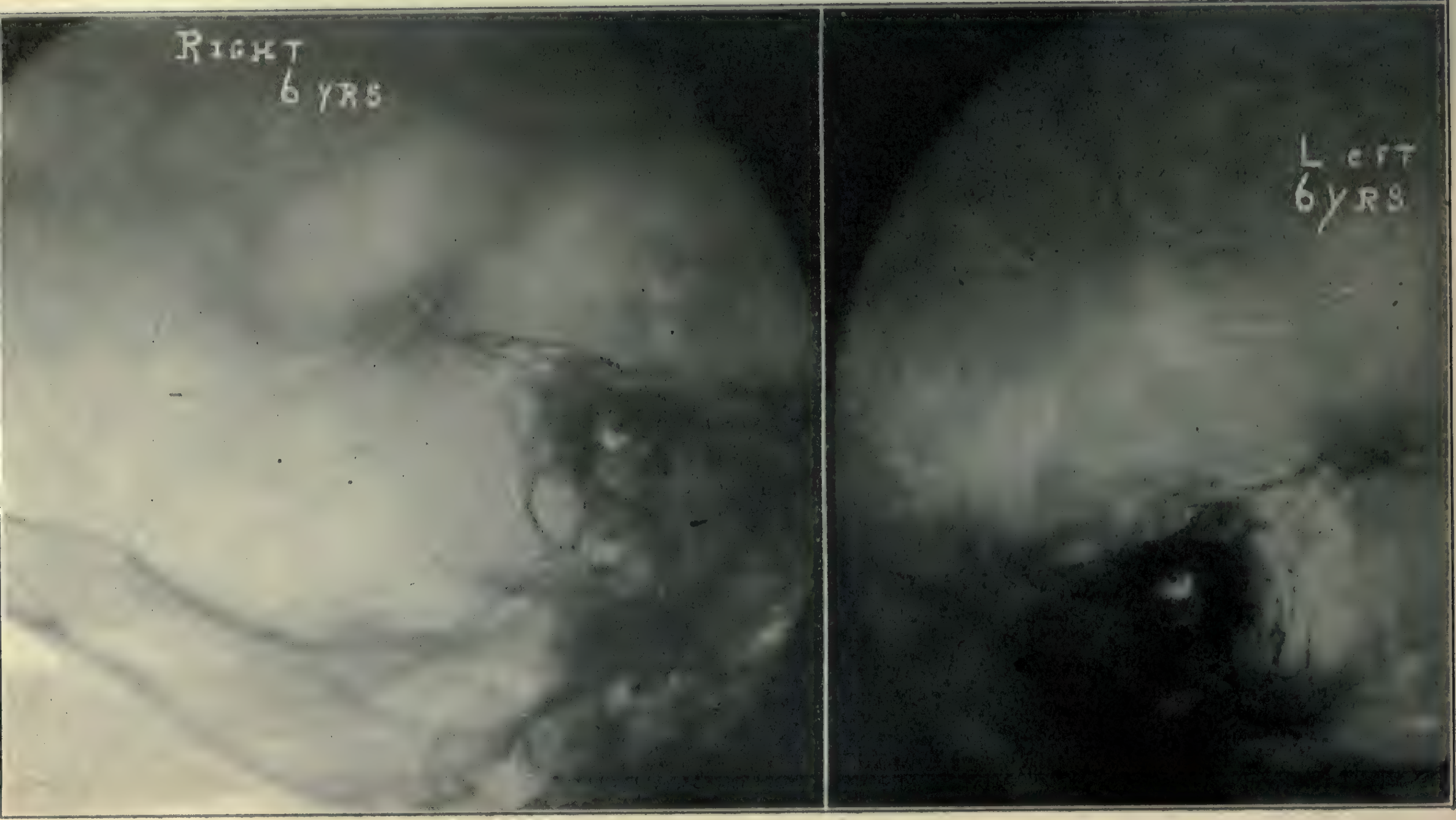
One of the difficulties we have to contend with is to procure skiagrams of uniform density of both mastoids. My observations are much the same as Lange's. He says: "As a rule, a slightly underexposed plate is best to study."

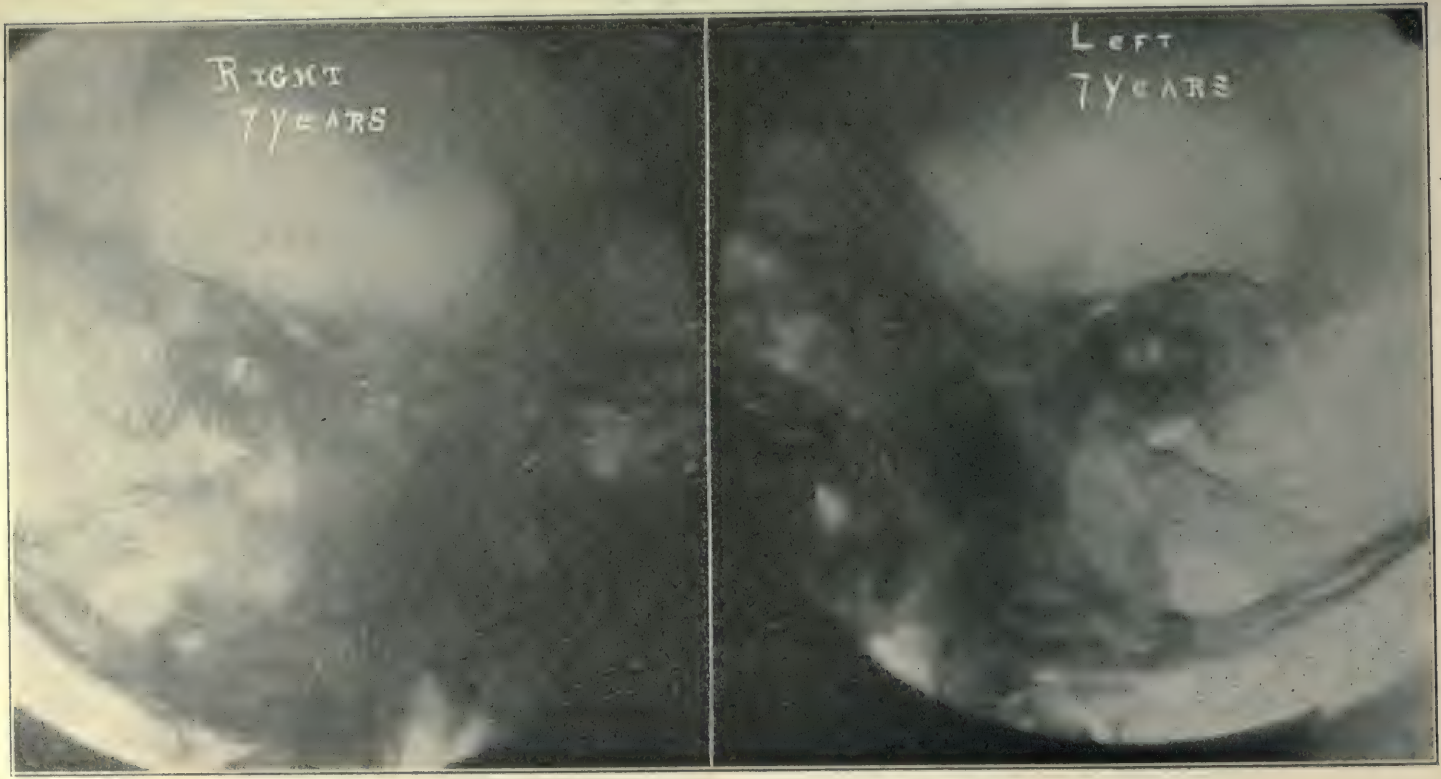
While examining numerous little patients with supposed fracture of the skull, in which a portion of the routine was radiographs of the occipital region, I frequently observed that the mastoid cells were well brought out in their transverse diameter and that the mastoid antrum was frequently seen. I have now adopted this as a part of my technic in mastoid cases—examining my patients first in this anteroposterior and then both sides in the posterolateral oblique positions.

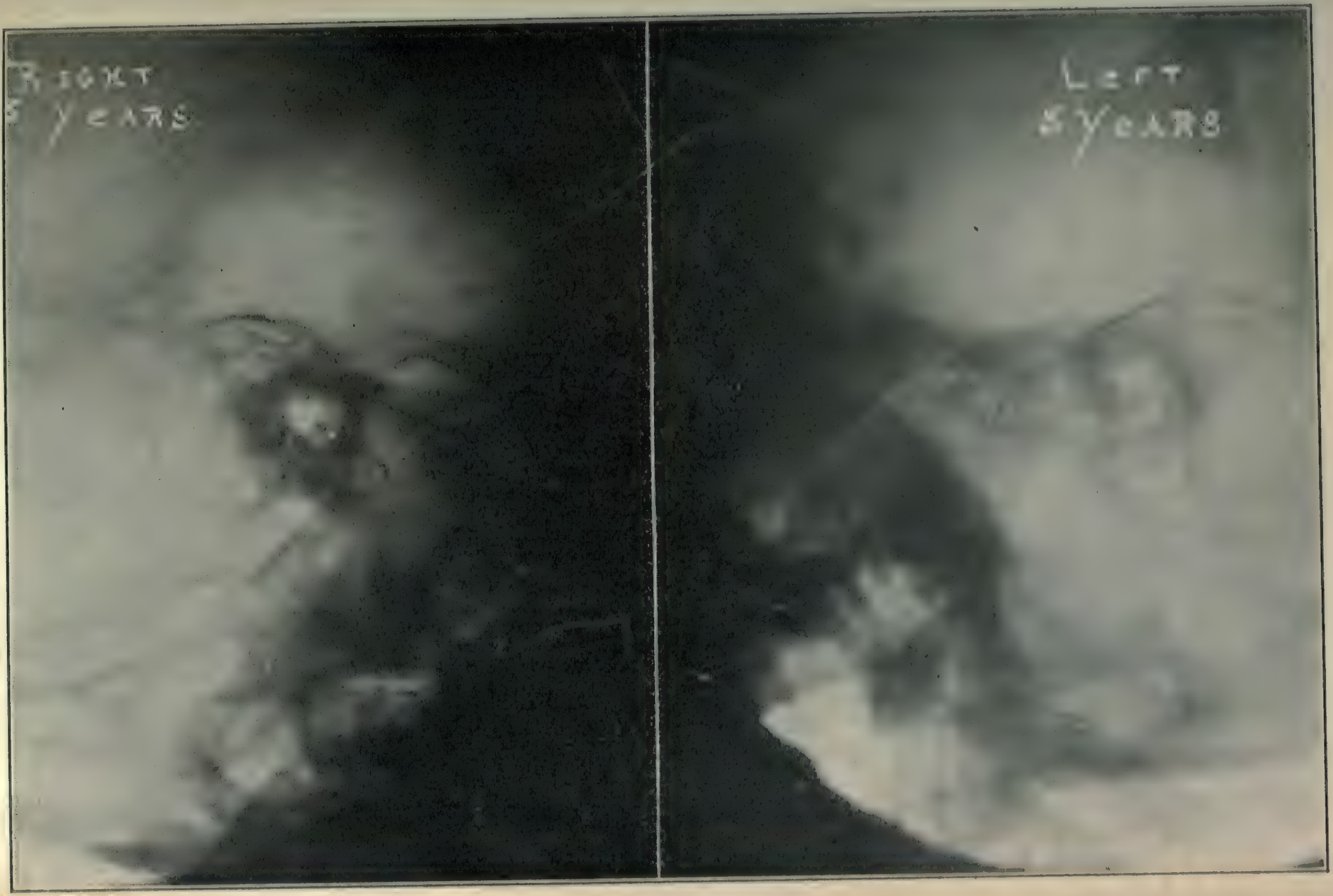

RIGHT
2 YEARS
LEFT
2 YEARS

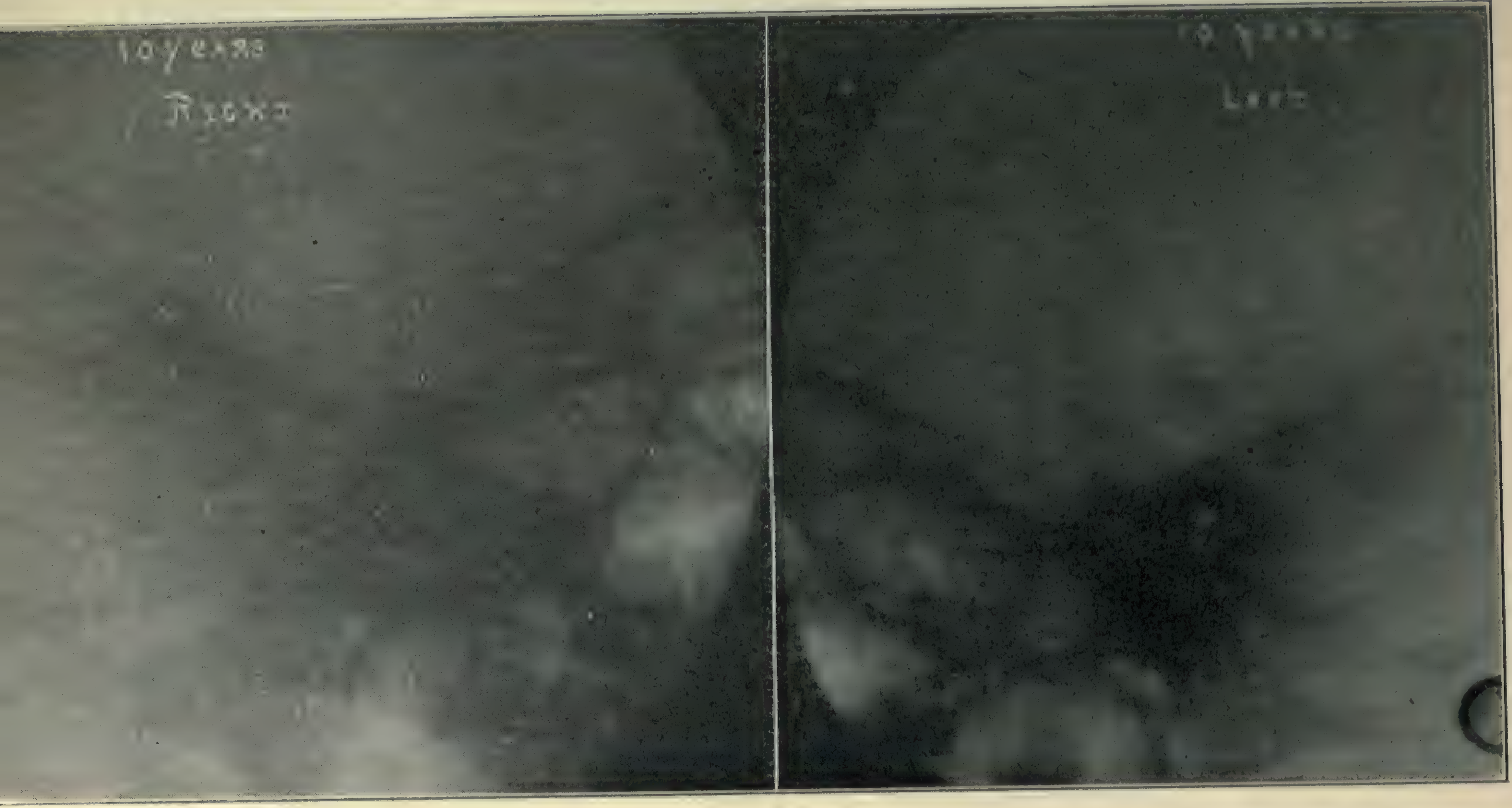

RIGHT
4 YEARS
LEFT
4 YEARS

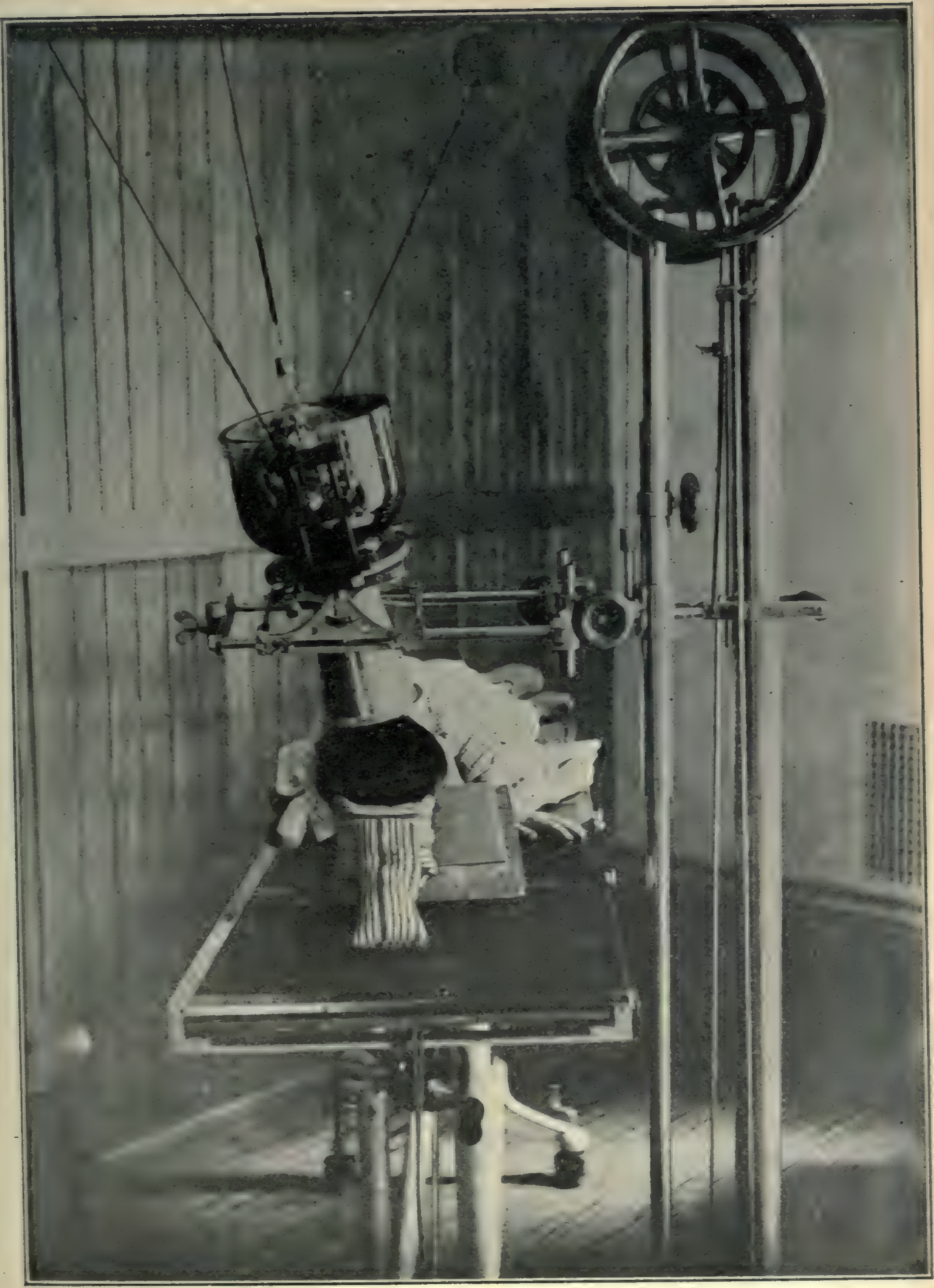



RIGHT
6 YRS
LEFT
6 YRS


Right
7 Years
Left
7 Years


RIGHT
5 YEARS
LEFT
5 YEARS


10 years
Right
10 years
Left



XLVII.

TOPOGRAPHY OF THE TYMPANIC CAVITY.*

By John A. Cavanaugh, M. D.,

Chicago.

The subject which is chosen for this thesis is one which most of our modern textbooks touch upon in a general way, and its importance is confirmed by personal observations on wet and dried specimens. Some of these specimens are photographed and are reproduced in this paper.

The development of the middle ear should be understood in order to know how the surrounding structures are sometimes invaded by diseases of this cavity; therefore, a brief résumé may not be amiss. The temporal bone is developed by ten centers, exclusive of those for the internal ear and the ossicles: one for the annulus, one for the squama zygomatici, two for the styloid process, four for the petrous, and two for the mastoid. Huxley declares the centers are more numerous and gives a somewhat different classification. The mastoid and petrous portions are united before birth; however, an imaginary line drawn in a vertical plane close behind the stylomastoid foramen and at right angles to the whole petromastoid portion, marks the union between these two portions. We have at birth three ununited portions of the temporal bone—the squama zygomatici, annulus and petromastoid parts—which are held together by bands of connective tissue. Ossification usually takes place between the two upper extremities of the annulus and adjacent portion of the external table of the squama, so that after a certain period the temporal bone separates into two portions—the united annulus and squamous portion and petromastoid portion. During the first year the bony union of these three parts, namely, the annulus, squama and petromastoid, are very imperfect. The annulus at birth (Plate I, Fig. 1) is like a plain ring, only that the upper posterior part is wanting, leaving a gap which is filled in by the outer table of the squama.

---

*Read before the Chicago Laryngological and Otological Society, February 18, 1913.

As a child develops there is an outward growth of the outer edge to form the future external auditory meatus (Plate I, Fig. 2-C and 2-A) and an inward growth from the inner edge to help form the floor of the middle ear. The horizontal portion of the squama divides into an outer and inner table, the outer forming the external wall of the attic, while the inner helps to form a part of the roof of the middle ear (Plate I, Fig. 3-A and B). The petrous portion forms the inner and posterior walls of the middle ear.

The tympanic cavity is the largest cavity of the temporal bone in the larger percentage of cases, but anomalies may exist where an unusually large antrum or mastoid cell is present. (Plate I, Fig. 6-A; Plate II, Fig. 10). I have in my collection a temporal bone wherein there is a cavity in the petrous portion extending from above the inner part of the internal auditory meatus, just internal to the superior and posterior surface, to the Glaserian fossa; it measures 20 mm. long, 9 mm. wide, and from 5 to 10 mm. deep (Plate I, Fig. 6-A). The tympanic cavity is of cuboid shape, the walls being very unequal. It is formed by the pars petrosa, pars squamosa and pars tympanica, though the pars petrosa forms the greater part. This cavity contains air, reduplications of mucous membrane, muscles, ossicles and the ligaments of the ossicles. This cavity we describe usually as having a floor, roof, an anterior, posterior, external and inner walls. If we were to consider the median plane of the skull, these are not quite accurate, for the inner wall lies at an angle of about 45 degrees from the sagittal plane.

The roof is concave downward from without in, and generally increases in height as it passes from the processus cochleariformis backward to become the roof of the antrum (Plate I, Fig. 5-A and B). It is formed mainly by a little lamina of bone which projects outward and forward from the superior surface of the petrous bone to join the inner table of the squama as it turns inward; this marks a fissure, the petrosquamous, which in children lies over the tympanum, but in adults is usually found a little external to this roof. It has no special anatomic features of interest, but it is of surgical and pathologic importance, since infection may pass through this area from the middle ear invading the meninges. Occasionally a dehiscence (Plate I, Fig. 4-A) exists, and the dura is in direct contact with the lining of the middle ear space. This fissure usually closes at the fifth month of life (Kopetzky). Examining 765 skulls, such defects were noted one hundred and sixty-seven times (Burkner). Körner found these dehiscences present eighteen times in 209 skulls, and also found in some cases the tegmen to be several millimeters thick, and where this exists, small air cells are adjacent to it. According to R. Johnson Held, the tegmen usually contains numerous foramina for the passage of small blood vessels, and at times has a cribriform appearance; he has found dehiscences twenty-nine times.

The floor is formed by a projection outward of the inferior surface of the petrous bone, which Gruber calls the fundus tympani, as it forms the greater part of the floor of the tympanum. This floor is also formed by the inward growth of the inner edge of the annulus. It narrows as the descending outer and inner walls approach each other. It lies a little below the floor of the external auditory meatus. It is pushed upward, forward and outward in some cases, while in others upward, forward and inward, at its posterior part, due to the encroachment of the jugular bulb (Plate I, Fig. 7-C, and Plate II, Fig. 8-F). There is sometimes a dehiscence in this area which allows the jugular bulb to be seen from the tympanum, and even gives a bluish tint to the lower part of the tympanic membrane in the living subject as seen through the speculum. Sohier Bryant, in the *Archives,* 1905, says: "In children the jugular fossa is below and not behind the tympanum." I have found both positions present. The bulb on the right side is usually larger, consequently there is more of an elevation of the floor, and, in my opinion, more reason for the presence of a dehiscence. I find, however, no literature verifying this idea.

The possibility of the existence of a dehiscence should always be remembered when performing a paracentesis of the drum membrane. In such conditions the bulb is always liable to become infected in suppurative conditions of the middle ear. Carl Koller, in *Archives,* 1904, reports such a case. Turgens (Warsaw) reports a hemorrhage from the bulb after the use of strong caustic in the middle ear. Lafarelle reports a case of a diverticulum of the tympanum from the floor, which extended downward, and was of the size of the tympanum, the walls of which were very irregular.

There is a small aperture in the floor, close to the inner wall, for the passage of Jacobson's nerve (Plate II, Fig. 8-C). In the macerated specimens this surface is usually very uneven, due to irregular cellular spaces.

The anterior wall can hardly be said to exist, for its place is taken up mostly by the tubal entrance and the tensor tympani muscle, the latter entering the tympanum above the tube (Plate I, Fig. 6-B and C). A small portion of the anterior wall does exist beneath the lower lip of the eustachian opening and floor of the tympanic cavity, where the fundus tympani turns up and separates the carotid artery from the middle ear. Sometimes this bony wall is wanting, thus allowing the carotid artery to be in contact with the lining of the middle ear. A number of cases are reported of fatal hemorrhages due to trauma of the anterior wall. Where a dehiscence is caused by an erosion, it is usually found at the vertical and horizontal part of the canal. Politzer attributes this to inflammatory softening due to pus which surrounds the artery, and which causes a diminished resistance; the repeated concussions of the blood wave finally results in a perforation of the anterior wall.

Another important anatomic feature of this anterior wall is around the lower lip of the tubal entrance, and consists of small cellular spaces known as Koerner cells (Plate II, Fig. 9-D), which become infected, and in some cases produce constant obstinate otorrhea. The failure to eradicate these cells thoroughly at the time of a radical operation is often the cause of constant trouble. Urbantschitsch, in the third edition of his textbook, speaks of finding in certain temporal bones cellular spaces in the vicinity of the tympanic orifice of the eustachian tube, which led to a system of pneumatic cells, and which in turn spread to the tip of the petrosal pyramid, and through dehiscences communicated with the middle fossa of the skull. I have one such specimen in my collection (Plate II, Fig. 10).

The posterior wall is absent in its upper part, due to a variable sized opening of a somewhat triangular shape, with its apex pointing downward (Plate II, Fig. 11-A). Its base is about 5 mm. wide, and from its base to the apex measures about 6 mm. The apex of this triangular opening lies just internal and above the upper end of the posterior arm of the annulus (Plate II, Fig. 12-E), and is called by some authors (Davis and MacEwen) the iter or aditus which leads into the mastoid antrum. Most of the literature I have consulted refer to a small recess just below the floor of this socalled accessory opening of the tympanum, the sella incudis, for the reception of the short process of the incus. This statement, according to my observation, is incorrect, as the short process rests on the floor of the iter or aditus close to the posterior wall, but not on the posterior wall. Close to the inner wall and below this opening is a small projection—the pyramid—with a tiny opening at its apex, from which issues the tendon of the stapedius muscle which arises from a cavity in its interior (Plate II, Fig. 13-D). From the inner and lower side of the pyramid I have found in most specimens a delicate thread-like bridge of bone extending to the upper edge of the beginning of the promontory. Below, at a point where the floor and posterior walls seem to meet, is a dense hard projection, which is the tympanic end of the styloid process. I have found in some cases, around this projection, numerous cellular spaces, which are important to remember in radical mastoid surgery (Plate III, Fig. 14-A). Through this wall passes the facial nerve, which enters just above and at the posterior end of the oval window, and runs in a somewhat diagonal direction, downward and outward, leaving this wall of the tympanic cavity just behind the middle of the posterior arm of the annulus (Plate I, Fig. 1-A; Plate II, Fig. 8-E, and Fig. 11-B). B. Alex. Randall, in the *Archives* of 1913, reports his findings in one hundred cases, where the facial nerve lay less than two nor more than four millimeters from the back wall of the meatus, and crossed the oblique plane of the drum three millimeters back of the middle of the posterior margin of the annulus. Hugh E. Jones, in the *Journal of Laryngology, Rhinology and Otology,* of June, 1903, and H. A. Alderton, in the *Archives* of 1904, give very important data regarding the location of this nerve. To avoid injuring this structure, one should not go below the floor of the antrum where it joins the posterior arm of the annulus (Plate II, Fig. 12-E).

The external wall is formed by bone and membrane, the upper part by the external table of the squama, which contains cellular spaces in the adult and lies over the superior meatal wall; the lower part by the drum membrane and annulus tympanicus (Plate I, Figs. 1 and 3). The lower edge of the external table of the squama for about four millimeters is not joined by the annulus (Plate I, Fig. 1-C, and Plate II, Fig. 12, between C and E). This area, which is in the upper and posterior part of the drum, and lies just anterior and slightly below the opening into the mastoid antrum, is where the periosteal covering of the upper part of the meatus passes into the inner surface of the external table of the squama, thus forming the periosteal covering of the outer wall of the antrum and attic, and allowing subperiosteal infections to travel into the external meatus from the attic and antrum at this point. Swelling here indicates attic and antrum inflammation. The annulus in most subjects is horse-shoe in shape, but cases are reported where the osseous circle is complete. Hyrtl has reported a case wherein two tympanic rings existed. The annulus forms only a small part of the external wall; on its concave side there is a sulcus—the sulcus tympanicus—for the insertion of the membrana propria (Plate I, Fig. 2-D). At the upper and posterior part, where the annulus is missing, the sulcus is also missing, and this area is known as the notch of Rivini (Plate I, Fig. 1-C, and Plate II, Fig. 12, between C and E). The annulus is placed obliquely, inclining toward the anterior wall and floor of the meatus, forming with the superior and posterior walls an angle of about 145 degrees, while with the floor and anterior wall it forms an angle of about 35 degrees. The annulus has a somewhat spiral shape, and its anterior extremity makes a slight twist on its long axis, so that the sulcus of the annulus is seen from the meatus in dried specimens when held in its normal position. At the upper end of this anterior arm is a small tubercle, the crista tympanica, and above this a little groove, the sulcus malleolaris, which lodges the processus longus of the malleus. The development of the annulus along the anterior part of the lower margin of the squama forms the Glaserian fissure, which is of special interest to the otologist, as it sometimes serves as a passage by which infection may spread from the tympanic cavity into the region of the parotid gland, or may even affect the inferior maxillary joint. This fissure is formed by the crista tympanica of the annulus and the tegmen tympani which advances to meet it; these structures draw nearer to the lower anterior margin of the squama and practically close this area; however, in the young, before this closure has taken place, and in those cases where development does not progress normally, it leaves a relatively large opening. Infection of the middle ear may travel through this opening and become the primary cause of some supposed parotid gland or pharyngeal abscess, and should be remembered.

The drum membrane is an irregular oval membrane, measuring about nine by ten millimeters (Plate III, Fig. 17). It is placed in the same plane as the annulus. The drum is concave outward at the umbo or tip of the malleus, and between that and the annulus it is convex outward, excepting in its posterior quadrant, where it is somewhat flattened. The membrane is transparent and about one-tenth millimeter in thickness. It separates the external auditory meatus from the middle ear. It consists of three separate layers: an outer cuticular, a central or membrana propria, and an inner or mucous layer. Zalewski, through experiments, found that pressure of from one to two atmospheres was necessary to cause rupture of the normal drum, which in atrophic conditions or cicatrices of the drum, infectious processes, or advanced age, lessened pressure was required. In fibrous or calcareous thickening, increased pressure was necessary. Gruber contends that the drum will stand a gradual increasing pressure of from sixty to seventy pounds.

There seems to be a diversity of opinion as to the position of the drum membrane. Most authors are inclined to believe the drum membrane lies more horizontal in the child than in the adult, while J. Pollak and Symington have agreed that the drum lies in the same plane in the adult as in the child. From my observations, I must agree with the latter (Plate III, Fig. 15 Adult and Fig. 16 Infant).

I believe that because of the high position of the auricle, the cartilaginous floor of the meatus lies almost parallel with the outer surface of the drum in children; while in the adult, because of the development of the osseous meatus, we look at the drum in a more horizontal direction. It would seem to me, if the annulus changed its position it would have some effect upon the size of the tympanic cavity, but this cavity is about the same in size in the young and old. I have removed the bony external auditory meatus in the adult to the drum membrane, and from all appearances it seems to be in the same plane as in a child. I have also made an artificial bony external meatus by casts and placed this against the infant annulus as it would appear later in the adult, and the drum seemed to be on the same plane as the adult membrane.

The membrana tympani is composed of a tense and a flaccid part. The former is tense because of the membrana propria, which is made up of the circular and radiating fibers, while the latter, or Shrapnell's membrane, is flaccid because it is devoid of these fibers (Plate III, Fig. 17-B). The long process of the malleus, of which we shall speak later, is readily seen through the drum membrane, and at the upper part of this handle is a round projection, the short process of the malleus. From the short process of the malleus, passing upward in opposite directions to the upper end of the annulus, is the edge of the membrana tensa, which is thickened and known as Prussak's fibers, called by some authors the anterior and posterior folds, while Bezold and Siebenman call them the anterior and posterior liminal strands (Plate III, Fig. 17-A and C). The area between these two bands is known as Shrapnell's membrane. It is of the greatest importance to remember the position of the drum membrane when removing foreign bodies from the meatus or performing a paracentesis. Perforations in the various parts of the drum are also of the utmost importance, and may be divided into the dangerous and nondangerous variety. When the membrane seems to be entirely destroyed in a marginal perforation at its junction with the annulus, or where there is a perforation in Shrapnell's membrane, or in the posterior superior quadrant, it is to be viewed with suspicion as of a possible dangerous type (Plate III, Fig. 19-A); while a perforation which exists where a membrane, be it ever so small in amount, separates it from the annulus, may be classified as belonging to the nondangerous variety (Plate III, Fig. 18-A). I am thoroughly convinced that in diseased conditions of the antrum, perforations at the posterior superior part of the drum and in Shrapnell's membrane are due to secretions drawing from the antrum directly upon the drum at this point, thus causing destruction in this area (Plate III, Fig. 20-A). The short arm of the incus forms with the outer table of the squama a trough, so to speak, which retains secretions and aids infections of Prussak's space (Plate IV, Fig. 23-A).

The inner wall looks forward and outward and is the most important of the various walls of the middle ear. It forms also the outer wall of the internal ear. From the tegmen tympani downward we observe:

First—A smooth bony projection in the upper posterior part, called the horizontal semicircular canal, which lies in a horizontal plane and passes back through the iter into the antrum (Plate I, Fig. 2-C).

Second—Below this elevation and lying in the same plane, but extending a little more anteriorly and not so far posteriorly, is another bony elevation, which marks the position of the canalis fallopii, through which the facial nerve passes (Plate II, Fig. 12-B). There is sometimes a dehiscence in this ridge, and mucous membrane only separates it from the tympanum. This is a very important surgical landmark, and its position should be thoroughly familiar to surgeons doing middle ear and mastoid operations. In infection of the middle ear where a dehiscence exists we are liable to meet with facial nerve complications, due to an exudate into the surrounding structures causing pressure. Infection may travel along the sheath of the nerve to the cranial cavity.

Third—Directly anterior and a little below where the facial canal makes its appearance on the inner wall is seen the processus cochleariformis, through which passes the tendon of the tensor tympani muscle (Plate I, Fig. 5-B).

Fourth—Below the canalis fallopii, near the posterior wall, is a recess at the bottom of which is an oval opening, the fenestra ovalis, measuring three millimeters long, and one and one-half millimeters wide, the long axis of which is directed upward and forward (Plate II, Fig. 8-A). In this oval opening rests the footplate of the stapes, which will be discussed later. This ossicle separates the vestibule of the labyrinth from the tympanic cavity. It is through this fenestra that most of the sound waves travel (Plate I, Fig. 3-D).

Fifth—Below this oval opening, helping to form its lower border and extending forward, is an elevation known as the promontory, which represents the first or beginning turn of the cochlea, and forms the greater part of the inner wall of the tympanic cavity (Plate II, Fig. 12-D). If we trace this prominence backward, we find it ends abruptly in a deep recess; the recessus rotundus. Opening forward from this recess in the macerated bone is a round aperture, the foramen rotundum, which leads into the beginning of the cochlea (Plate IV, Fig. 21-A). In the recent state this opening is closed by a membrane, the membrana secundaria, which regulates the fluid waves that have passed through the cochlea. Opinions differ as to whether sounds travel through the fenestra rotunda from the middle ear. According to Gruber, this round window lies nearly parallel to the drum membrane in the fetus at the third or fourth month, while at birth it lies obliquely to the membrane, and as the child advances in years this opening gradually changes until it looks toward the posterior wall. Troltsch is of the opinion that the infantile position of the round fenestra may under certain conditions persist. This round opening varies from one and six-tenths to three millimeters in height and its width from one to three millimeters. The membrane which closes this round opening is somewhat convex toward the interior of the cochlea. It is covered on the cochlea side by the labyrinthine lining, and on the tympanic side by the mucosa of the lining of the middle ear.

The tympanic cavity measures through its horizontal plane ten to thirteen millimeters; its vertical plane from eight to fifteen millimeters. The roof increases in height from the anterior part of the cavity to the posterior, where it continues on as the covering of the antrum. The width of this cavity varies; at the umbo and promontory it is about two millimeters, and from that the width varies up to seven millimeters. The quantity of fluid which this cavity will hold is about ten minims in the recent specimen, and in the dried about twelve minims.

This cavity is divided into three chambers by imaginary lines: the upper or attic, the middle or atrium, and the lower or hypotympanic space (Plate III, Fig. 20-D).

The attic is that portion lying above an imaginary line drawn from the prominence of the horizontal portion of the facial ridge to the short process of the malleus. Some authors draw the line to the lower edge of the external table of the squama or upper edge of Shrapnell's membrane. Kretschman and

Siebenman contend the former should be adhered to, and base their opinion on the clinical fact that it is a common observation to find the membrana flaccida involved when the pathologic process is limited to the attic. The attic lies almost directly above the posterior quadrant of the drum membrane (when the head is held in its horizontal plane) and lower ledge of the outer table of the squama. If a perpendicular line were drawn from the inner wall of the attic downward, it would pass through the drum membrane near its upper edge and strike the floor of the external meatus about three millimeters external to the lowest edge of the drum. If drawn in like manner from the extreme outer part of the outer wall, it would strike the floor of the meatus about seven millimeters from the lowest edge of the drum. To this space Bezold, Troltsch and Siebenman have given the name aditus ad antrum; Schwalbe, cavum epitympanicum; Liedy, attic, and Hartmann, the cupola. In this socalled chamber rests the heads of the malleus and incus and their ligaments (Plate IV, Fig. 23-A). The book of Surgical Anatomy by Davis and the book by MacEwen, describe the attic containing the above structures, but continue to say that the short process of the incus projects into a space called the iter or aditus, and that through this space the attic communicates with the mastoid antrum. They describe the space as being a quarter of an inch long from the posterior extremity of the attic to the antrum, and that it is covered by the tegmen, the inner and lower wall by hard bone for the seventh nerve and external semicircular canal. This chamber is of the greatest importance because of the many structures it contains and because of its close association with the cranial cavity and mastoid antrum. Because of the number of structures and reduplication of mucous membrane, infection of this region is arduous to master on account of the difficulty in establishing drainage (Plate IV, Fig. 25-C). The incus aids us in many cases; because of its position, it acts as a natural drain from this region. The attic is again divided into an internal and an external chamber. The area lying between the malleoincudal articulation and the external table of the squama and Shrapnell's membrane is called the external attic; that part internal to these structures, the internal attic. This external attic is again subdivided into an upper and lower space. The lower one is known as Prussak's, and is bounded internally by the neck of the malleus, inferiorly by the processus brevis, externally by Shrapnell's membrane, and superiorly by the external ligament of the malleus. This space measures about one and a half millimeters high and two millimeters at its base. According to Politzer, this space opens occasionally into the anterior and posterior pouch of Troltsch. From my observations of work in the attic region, I have come to the conclusion that the number of trabeculæ in the attic varies in many cases, and I think it due to the process of development.

The atrium is that part of the tympanum lying opposite to the drum, below the imaginary floor of the attic and above a plane on a level with the floor of the external auditory meatus. In this chamber are found the handle of the malleus and the long process of the incus, stapes, oval, round window and opening into the eustachian tube.

The hypotympanic space lies below the lowest edge of the drum membrane or below the floor of the external auditory meatus, and contains a variable number of cellular spaces.

The ossicles of the middle ear are three: malleus, incus, and stapes.

The malleus is club shape, measures about nine millimeters in length and weighs about twenty-two milligrammes. It is made up of a head, neck and handle. The head and neck form with the handle an angle of about 150 degrees. The head is irregularly globular in shape, and its upper part lies about one millimeter from the tegmen. It has a free convex surface, and on its inner and posterior surface has an oval depression for articulation with the incus. The neck is a fraction over one millimeter long, and extending forward from its anterior surface is a delicate spicule of bone, the processus gracilis, the remains of Meckel's cartilage. Extending in an obtuse angle from the neck, downward and inward, is the handle, which is prismatic on cross sections, and tapers gradually from just below the neck to the tip, which is turned slightly forward and somewhat flattened. The handle measures five millimeters from short process. At the upper end of the handle is a somewhat rounded point, projecting against the drum, and called the short process. On examining the handle while connected with the drum, the inner two-thirds of its circumference seems to be free toward the tympanicum, excepting for the mucous membrane covering, while the outer one-third seems imbedded in the membrane (Plate III, Fig. 20-B). The membrane can be easily separated from the malleus with the exception of its lower end, where it is firmly attached. Gruber contends that a cartilaginous structure surrounds the malleus where it is imbedded in the drum, and the bony part rests in this cartilaginous bed. This ossicle, especially the head and short process, is practically always preserved in suppurative conditions, because of its rich blood supply, which will be spoken of later. A case of congenital gap in the handle of the malleus has been reported by A. H. Cheatle. The ligaments which hold this ossicle in place are: the suspensory, a short bundle of fibers attached to the upper surface of the head and tegmen tympani; it checks the outward movements of the handle. The external is a fan-shaped structure attached to the neck of the malleus and diverging as it passes outward to become attached to the lower edge of the outer wall of the squama at the Rivinian notch. This ligament forms the roof of Prussak's space. There is a small bundle of fibers at the posterior part of this ligament which is separated from the main bundle, and called by some authors the posterior ligament of the malleus. The anterior ligament leaves the neck of the malleus around the processus gracilis, encircling this process and passing to the Glaserian fissure and anterior spine of the tympanic ring. The capsular ligament surrounds the malleoincudal articulation, and holds these surfaces together. Because of the number of ligaments which are attached to this ossicle, it makes it firm and secures it in one place, therefore, making it a valuable landmark.

The incus, called so because of its shape, has a body, a long and short process. On the anterior surface of the body is a saddle shaped depression, for the articulation of the head of the malleus. The two surfaces of the malleus and incus are so arranged that when the drum membrane is pushed inward by a sound wave, a small ridge on the malleus fits into a depression on the incus, thereby bringing pressure on the stirrup, while if the drum is pushed outward by inflating, etc., these two surfaces separate and the malleus moves outward without pulling the incus with it, thus preventing the dislocation of the stapes from the oval window during inflation, etc. The short process measures three millimeters, and in some cases a fraction over; the long process, from the lower edge of the articular surface to the tip, about four and a half millimeters. This ossicle weighs about twenty-four milligrammes. The short process is directed backward and is lodged in the sella incudis of the posterior tympanic wall, just below the opening into the antrum mastoideum. The long process, downward and inward, parallel and posterior to the malleus handle. At its lower end it turns at a right angle inward to join the head of the stapes. The right angle projection is known as the processus lenticularis or orbicularis. In the fetus there is a separate bone (Quain). In inflammatory conditions of the tympanum the incus is the first of the ossicles to be thrown off and destroyed because of its limited blood supply, and because of the position of the artery which supplies it, which is so readily pressed upon by the swelling of the tissues in that area. The ligaments which secure this ossicle in position are: the capsular, already spoken of; the ligament of the incus which surrounds the short process, passes back and is fastened to the area of the floor of the iter where the short process is lodged. There is a small ligament which holds the os orbicularis and head of the stapes together.

The stapes consists of a head, two crura and a footplate. The head is directed outward and articulates with the processus lenticularis of the incus. It is disputed as to whether this is a synovial joint, but Rudinger describes it as such. There is a short neck which divides into two crura, the anterior and posterior, which are united to a flat oval plate, the footplate. The footplate occupies the greater part of the fenestra ovalis, but it is in no place in actual contact with the bone, there being a layer of cartilage all around the footplate, and another around the edge of the oval window. Between these two layers of cartilage is a ligament. The length of the footplate is a fraction over three millimeters, its width one and a half millimeters. The anterior crus measures about two millimeters, its posterior crus three millimeters. This ossicle weighs about two milligrammes. The ligaments are the annular, which holds the footplate in the oval window, and one which unites the head with the incus.

The muscles of tympanum are two: the tensor tympani and the stapedius. The tensor tympani arises from the cartilaginous portion of the eustachian tube, small area of the great wing of the sphenoid around the tube, and from the walls of the bony canal which lodges the muscle. It is a little over half an inch long. Near the opening of this tube the muscle becomes tendinous, passes through the processus cochleariformis at right angles to its belly, passes outward across the tympanum to be inserted into the inner surface of the manubrium of the malleus, where it joins the neck. Gruber says the tendon is attached to the inner margin and anterior surface of the malleus.

The stapedius muscle arises from a canal in the posterior wall of the tympanum, lying parallel with the fallopian canal, but a little anterior and internal to it. It emerges through a small opening, the pyramid, to be inserted into the head of the stapes. Rudinger says it is inserted into the lower extremity of the descending process of the incus. In most of the cases which I examined the muscle seemed to vary in its insertion; sometimes it would be attached to the posterior crus, again to the head and posterior crus, then again some of the fibers seemed to extend from the head to the os orbicularis, but most often to the neck.

Various theories are advanced regarding the function of these muscles, but they are beyond the scope of this paper.

The arterial supply to the tympanum is the tympanic branch of the internal maxillary, which enters this cavity by way of the Glaserian fissure; it supplies the anterior part of the cavity, and its branches anastomose along the periphery of the drum membrane with the stylomastoid branch of the posterior auricular. This tympanic branch sends branches into the external auditory meatus, and is called the deep auricular.

The tympanic branch of the internal carotid passes through the anterior wall of the tympanum into the middle ear. The stylomastoid branch of the posterior auricular passes up the fallopian canal with the facial nerve and sends branches through the posterior tympanic wall to the tympanum and drum membrane.

The petrosal branch of the middle meningeal, which anastomoses with the stylomastoid in the fallopian canal, sends branches into the tympanum. Zuckerkandl mentions a small branch of the stylomastoid artery, which he found constantly passing out from the middle part of the fallopian canal to supply the stapes and membrana obturatoria, then to the promontory to anastomose with the above branches, which he calls arteria stapedia.

The tympanic branch of the ascending pharyngeal, Gray says, ascends through the eustachian tube, while Deaver speaks of its entrance through the tympanic canaliculus with Jacobson's nerve. All these branches which supply the tympanum anastomose freely. However, Prussak states that the arteries of the tympanic cavity have an extensive course without anastomosing with one another. He says they divide at an acute angle into branches the caliber of which are large in comparison to the parent trunk, these branches passing almost at once into small veins, capillaries in many parts being absent.

Because of the free passage of the arterial supply through the periosteum to the subjacent bone, inflammatory processes of this cavity have a great tendency to involvement of the osseous wall.

There is considerable controversy as to whether the arteries of the middle ear anastomose with the arteries of the labyrinth. Politzer describes such branches existing. Shambaugh, who has studied this phase of the subject very thoroughly, has found that there is an anastomosis existing between these two chambers. (*Archives of Otology,* 1906.)

The venous blood of the tympanum is carried off by the temporomaxillary vein, superior petrosal sinus, lateral sinus, internal jugular, and pharyngeal veins.

Numerous small vessels communicate with the dura through the tegmen tympani. Inflammatory processes may travel by these veins to the various areas through which the veins travel and are, therefore, of pathologic importance.

The lymphatic distribution of the tympanum is not well known, but according to Rauber they accompany the blood vessels. There is a close connection with the prevertebral glands, which should be remembered in a retropharyngeal abscess, during middle ear suppuration.

The nerves of the tympanum are derived from several sources. The relation of the seventh nerve to the tympanum has already been considered. The nerve supply of the middle ear is derived from branches of the fifth, seventh and ninth cranial nerves, which form a plexus on the promontory known as the tympanic plexus. The branches which form this plexus are Jacobson's nerve from the ninth, which passes through the canaliculus of the floor of the tympanum just below the promontory. The small deep petrosal from the carotid plexus of the sympathetic, which enters through the anterior wall. The small superficial petrosal branch of the seventh, which enters the tympanum beneath the tensor tympani canal. The great superficial petrosal, another branch of the seventh, which enters the tympanum just anterior to the oval window.

The chorda tympani nerve passes through the tympanic cavity, entering at the upper posterior arm of the annulus through a small opening, the iter chordæ posterior, passes forward between the long process of the incus and neck of the malleus, just above the junction of the tensor tympani muscle with the malleus, and passes out of the cavity through a small opening at the upper anterior arm of the annulus, the iter chordæ anterior (Plate II, Fig. 12-C, and Plate IV, Fig. 22-A). The position of this nerve in the tympanum causes two pockets to be formed, one anterior and the other posterior to the malleus, and known as the anterior and posterior pouches of von Troltsch (Plate IV, Fig. 24). The mucous membrane passes down from the roof of the tympanum, covers over the chorda tympani nerve, then passes up again to be reflected into the drum membrane; therefore, these pouches open downward and lie between this fold of mucous membrane which covers the chorda tympani and the drum membrane. The posterior pouch is the larger of the two. Troltsch considers these folds of mucous membrane as true duplicatures of the membrana tympani, and has found fibers in them similar to those in the drum. Gruber, after the most careful study, has never been able to demonstrate the fibers of the membrana propria, therefore, he considers them folds of mucous membrane. Rudinger regards them as forming bands of attachment for the upper part of the malleus.

Politzer says there are openings in some cases into these pockets from Prussak's space. I have followed his method of experimenting by putting quicksilver into the pouches and opening Prussak's space in eight cases, but the quicksilver did not leave the sacs. I also examined these pouches under high magnifying power, but could see no such openings.

The nerve supply of the drum membrane has been carefully studied by J. Gordon Wilson, who says: "The drum of man is chiefly supplied by nerves which enter from the external auditory meatus. They pass in as one large trunk at the upper posterior part of the auditory canal. Numerous smaller branches enter from the periphery. There are but few nerves entering this structure from the tympanum. These branches are derived from the auricular temporalis and vagus."

I am greatly indebted to Dr. Joseph Beck for many suggestions which were of value to me in the presentation of this subject.

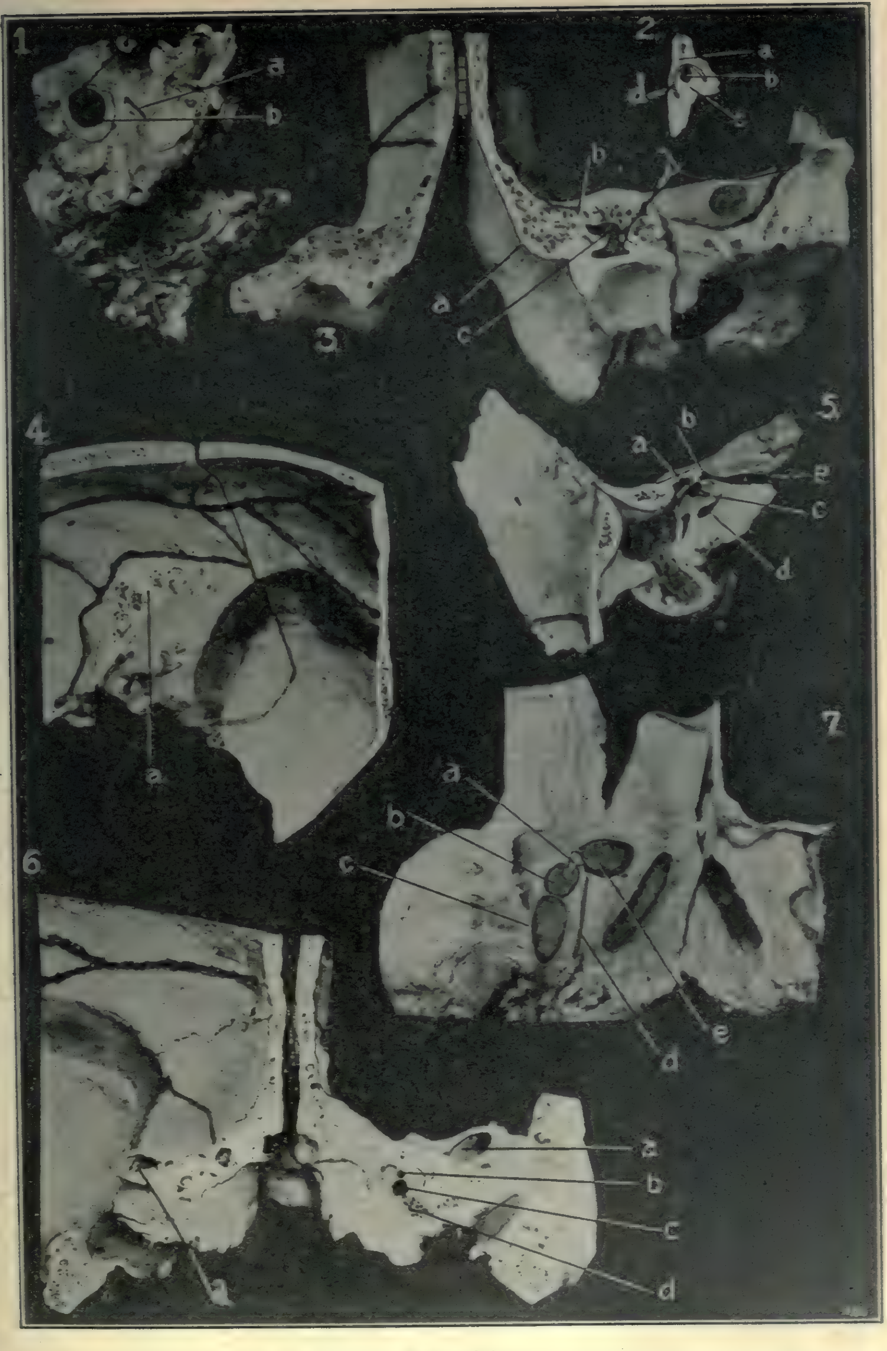


PLATE I.

FIGURE 1

FIGURE 10—a—Inner ear.
FIGURE 11.—a—Mastoid antrum. b—Facial nerve.

nal. c—Drum and long process of the incus. d—Space in the bone for the stapedius muscle.

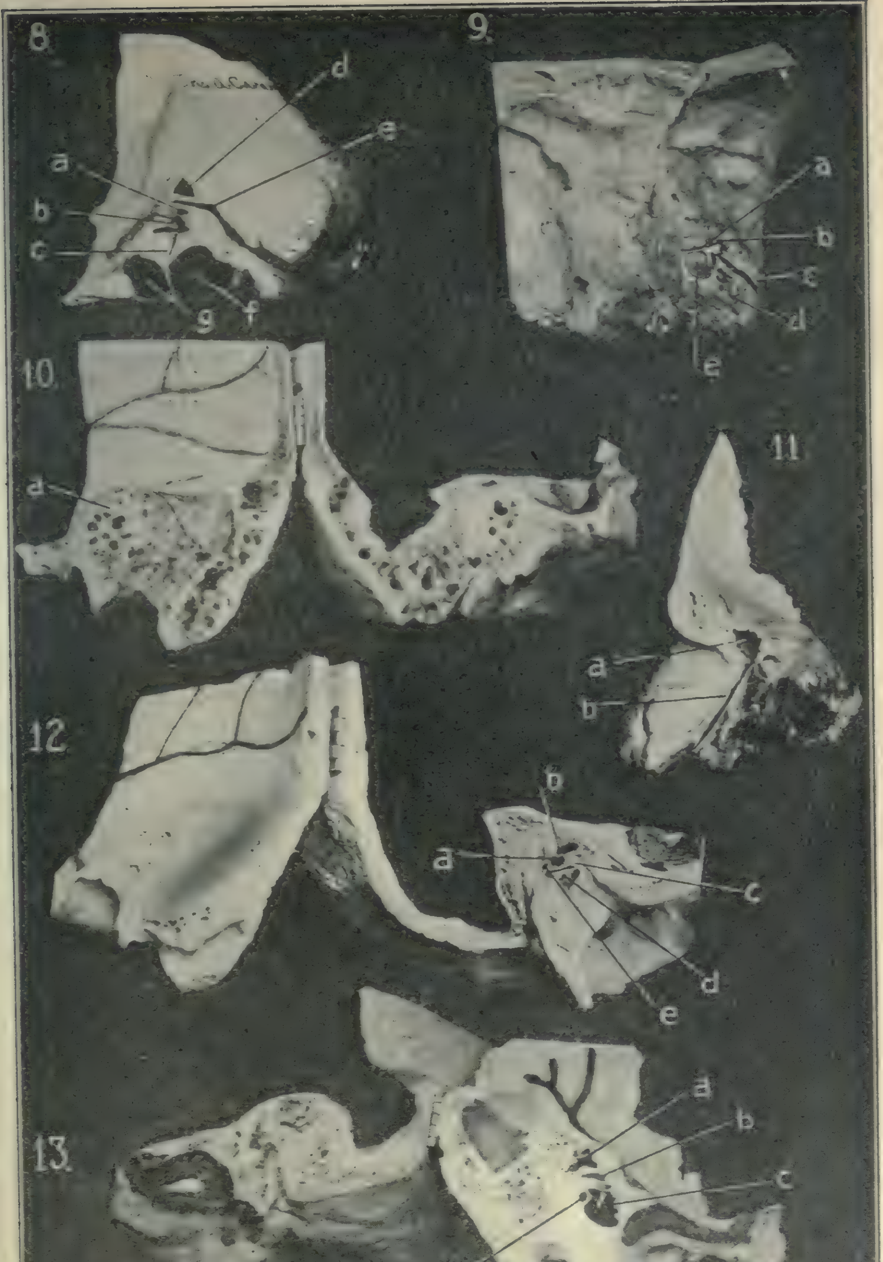


PLATE II.

FIGURE 8—a—Oval window. b—Promontory. c—Jacobson's or tympanic nerve. d—Opening into mastoid antrum. e—Facial nerve. f—Jugular bulb. g—Internal carotid artery.

FIGURE 9.—a—Chorda tympani nerve. b—Attic. c—Eustachian tube. d—Koerner cells around tubal opening. e—Drum membrane.

FIGURE 10—a—Inner ear.

FIGURE 11.—a—Mastoid antrum. b—Facial nerve.

FIGURE 12—a—Oval window. b—Facial nerve in inner wall of middle ear. c—Anterior arm of the annulus. d—Chorda tympani nerve. e—Posterior arm of the annulus.

FIGURE 13.—View of the middle ear from the inner side. a—Mastoid antrum. b—External semicircular canal. c—Drum and long process of the incus. d—Space in the bone for the stapedius muscle.

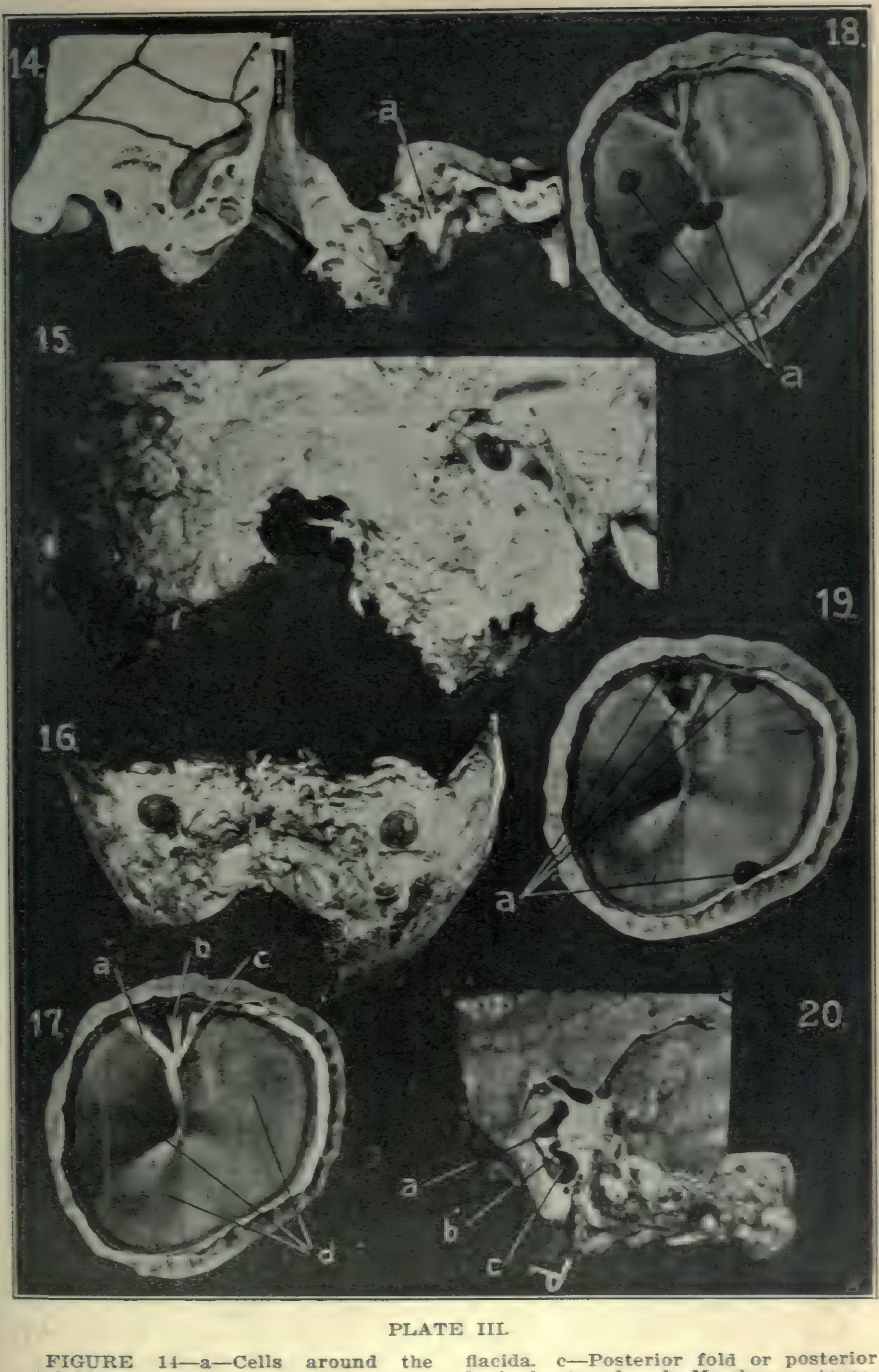


PLATE III.

FIGURE 14—a—Cells around the styloid process.

FIGURE 15.—The adult drum membrane, with the bony and cartilaginous parts of the meatus removed as far as the annulus.

FIGURE 16.—Infant drum membrane and annulus at birth.

FIGURE 17.—(Deaver.) a—Anterior fold or anterior liminal strand. b—Shrapnell's membrane or membrana flacida. c—Posterior fold or posterior liminal strand. d—Membrana tensa.

FIGURE 18—a—The nondangerous variety of perforations of the drum membrane.

FIGURE 19—a—The dangerous variety of perforations of the drum membrane.

FIGURE 20.—a—The attic leading to the antrum. b—The incus. c—Drum membrane. d—Hypotympanic space.

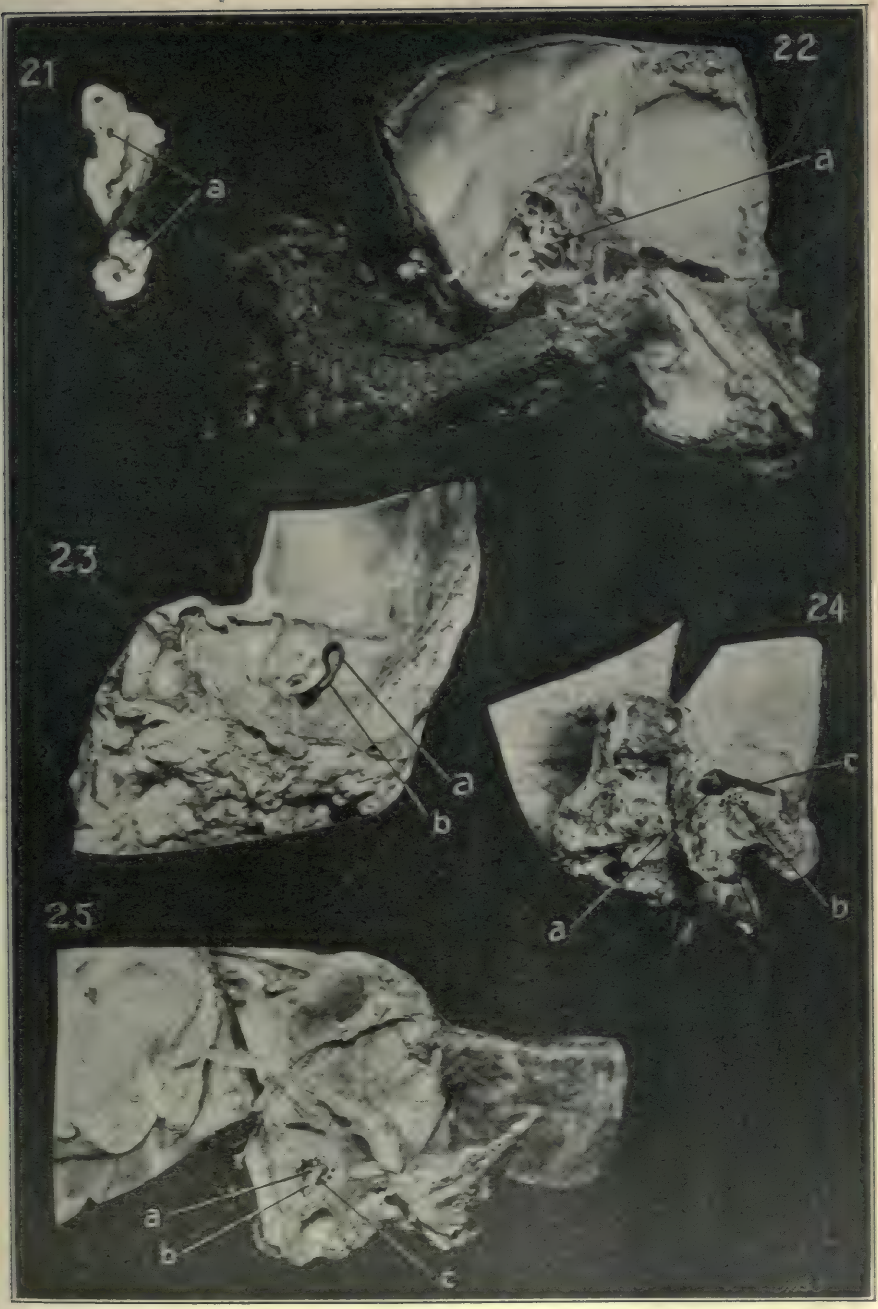


PLATE IV.

FIGURE 21.—a—The round windows.

FIGURE 22.—Outer wall of the middle ear, viewed from within; showing opening into the antrum, attic, ossicles, and drum membrane.

FIGURE 23.—a—Attic. b—Atrium. Malleus in position.

FIGURE 24.—a—Posterior pouch of Troltsch. b—Koerner cells around opening of eustachian tube. c—Eustachian tube.

FIGURE 25.—Middle ear from above, with the tegmen tympani and antri removed. a—Head of malleus. b—Short process of the incus. c—Fibrous bands between heads of the ossicles and external table of the squama.

XLVIII.

SOME ANATOMIC AND CLINICAL RELATIONS OF THE SPHENOID SINUS TO THE CAVERNOUS SINUS AND THE THIRD, FOURTH, FIFTH, SIXTH AND VIDIAN NERVES.*

By Greenfield Sluder, M. D.,

St. Louis.

The body of the sphenoid bone is usually hollowed out by the sphenoidal sinus. This cell may, however (rarely), be rudimentary and occupy a very small space in the lower anterior part of the body, which is otherwise hollowed out by a postethmoidal cell. It is of the cell which hollows the body of the sphenoid that I speak, regardless of whether it be the sphenoidal sinus proper or a postethmoidal cell.

The body of the sphenoid is covered above and laterally by the dura mater, with the cavernous sinus between its external and internal surfaces (in it), occupying a position for the most part above and lateral to the body. Within the cavernous sinus are found the internal carotid artery and the third, fourth and sixth cranial nerves, with the first division of the fifth lying in the lower part of its lateral wall. The impression given in the treatises on anatomy is, usually, that these nerves are rather widely separated from the sphenoid sinus, as shown in Figure 1. The second and third divisions of the fifth and the Vidian are also usually represented as well removed from this cell—that is, separated by a considerable thickness of bone. (Fig. 1.) The fact is, the sixth and the third division of the fifth are the only ones of these nerves that are not at times in close association with this cell—that is, separated from it by a very thin layer of bone; and even the third division of the fifth is sometimes also in rather close association with it. (Figs. 2i, 2ii.) The sixth, so far as I have found, is uniformly placed on the lateral aspect of the carotid and always removed from this bony wall. Thus far I have been speaking of a sphenoid sinus which is limited to the body of the sphenoid. Should it be extended into the greater wings, the sixth nerve may come into close association with it in the sphenoidal fissure below.

---

*Read before the American Laryngological Association, Atlantic City, N. J., May 11, 1912.

The fact which determines the relations of these nerve trunks to the sphenoid sinus is the size of the cavernous sinus rather more than the size of the sphenoid sinus. If the cavernous sinus be large in length and cross section, these nerve trunks will usually be far removed from contact with a sphenoid sinus of any size. On the other hand, if the cavernous sinus be small in length or cross section, these nerve trunks may be closely associated with a sphenoid sinus limited to the body of the sphenoid bone. Of course, a small sphenoid sinus in the center of the body of the sphenoid bone will be widely removed from contact with these nerve trunks, regardless of whether the cavernous sinus be large or small. A sphenoid sinus of large extent—prolonged backward and outward—may readily closely approach the third division of the fifth in the foramen ovale (Figs. 2i, 2ii), and Dr. H. P. Mosher has shown me two specimens in which the sinus extended to a close association with the Gasserian ganglion. In these two specimens the internal carotid rising out of the foramen lacerum medium was surrounded on the internal half of its circumference by the sphenoidal sinus, the wall of which was less than the thickness of a hen's egg shell at this place (Fig. 4ii.) A sinus extended downward soon approaches the Vidian nerve. In fact, the upper side of the bony case of the Vidian canal is often partly deficient under these circumstances. (Figs. 2ii, 3, 4.) The excavation may also extend below the level of the canal and leave it stalking through the sphenoid sinus like an ancient aqueduct, over the plain, connected with the bone below by a paper-like support. (Fig. 3.) Or it may be protected on one side of, and exposed on the floor of the sinus of the other side of the skull. (Fig. 4.) The sphenoid sinus extended laterally soon reaches the foramen rotundum, and may then environ one-half of its circumference, bringing the second division of the fifth at this point, and for some distance posterior to it, into very close association with it. (Fig. 5.) The third and fourth nerves are in close association with the anterior clinoid process or lesser wing of the sphenoid, which are not infrequently hollowed out by a prolongation of the sphenoid sinus or (from in front) by an extension from a postethmoidal cell, thereby bringing these nerves also into close association with these cells. (Fig. 6.) The first division of the fifth comes into close association with the sphenoid sinus anteriorly, if the cavernous sinus be small in either direction. (Figs. 7, 8.)

Clinically the inflammatory diseases of the accessory sinuses of the nose vary greatly. Each has, however, some semblance of constancy in its behavior, at least as far as symptoms with signs go, with the exception of the sphenoid. As is known to us, it is no uncommon thing to see a sphenoidal empyema give rise not only to its own characteristic symptoms, but to simulate the pains produced by all the other sinuses. Pain of a frontal sinus inflammation may be simulated by a sphenoidal inflammation—the differential diagnosis being made by the absence of pus from the frontal, with no tenderness of its floor, as well as a negative finding on the Roentgen ray picture. The pain of a maxillary antrum inflammation may be differentiated by a negative antrum puncture as well as a negative Roentgen ray picture. Absence of pus in or from the ethmoid would exclude the ethmoidal cells. These symptoms, when produced by the sphenoid, are, however, more apt to be nocturnal than diurnal.

It seems to me there are two pictures produced by inflammatory disease of the sphenoid sinus. The primary simple one is that produced by the pressure from an obstructed outflow of pus. I have construed this as the explanation of the somewhat "dull," "heavy" pain in the back of the head described by these patients. The more complex one, simulating the pains which are ordinarily produced by the other sinus, can only be explained by assuming that the associated nerve trunks have become involved either by the inflammatory process or by its toxins. This assumption applied to the third nerve explains also the dilated pupil seen in some of these cases, when the ophthalmologist or neurologist can assign no reason for it; and the asthenopia that is sometimes met with when no reason may be found in the eye or nose, otherwise. Some of these cases show a paresis of the superior oblique.

From the above sketched anatomy it would seem possible with the arrangements cited, by which the sphenoid sinus is separated from the nerve trunks by thin divisions of bone, that such an extension of inflammation might easily occur. In an effort to prove the accessibility of these nerve trunks I have ten times painted the cavity of the sphenoid in a stripe from above downward and outward with a very small applicator bearing about one-half drop saturated water solution of cocain and paralyzed the three divisions of the fifth for tactile sensation and pain, accompanied by a marked sense of stiffness of the lower jaw on that side. A needle passed through the skin supplied by these nerves failed to evoke sensation. In these cases the separating wall must have been a very thin one, thereby permitting the cocain to pass quickly and easily through to the nerve trunks. I have also, for therapeutic purposes, filled the sphenoid with oil solutions—1 per cent phenol, 2 per cent menthol, 5 per cent and 10 per cent oil wintergreen and 1 per cent cocain alkaloid—and found that they all produce a well defined analgesia of the first and second and often the third division of the fifth, accompanied by very little or no tactile anesthesia. It therefore seems to me reasonable to assume, when the diagnosis is sphenoid empyema and the symptom is pain in the brow, that it is inflammation or irritation of the first division of the fifth by the process within the sinus which makes the symptom. When the symptom is pain in the upper jaw and teeth, it seems reasonable to assume the second division of the fifth has become involved. When the symptom is pain in the lower jaw and teeth, accompanied by a sense of stiffness in the jaw on that side, it may be assumed that the third division has become involved and that probably in the foramen ovale.

The behavior of certain sphenoid empyemata has interested me much for several (five) years. These are cases in which the pains and aches have continued unchanged, despite wide opening of the cavities and cessation of all pus or signs of local disease. It is my habit to perform the Hajek radical postethmoidal sphenoidal operation. It seems to me the most comprehensive treatment. Some of these cases, however, although recovering from the suppurative inflammation, and later from all signs and symptoms, have frequently subsequently developed headache and behaved as cases of migrain. Dr. M. A. Bliss has seen a number of these cases in consultation, and has sent some of them to me and has agreed in the diagnosis of migrain. We have felt that in at least this class of cases which have simulated and borne the name of migrain, that the origin of the pain was a local one. These are of similar symptom complex to those of toxic origin. The distribution of the pain has been that suggesting as its origin the first division of the fifth, as, or well as, the second division, sometimes, or the third division, and combined at times with the posterior pain, which I have attributed to the irritation of the Vidian. In a recent paper[1] I mentioned that in the injection of the sphenopalatine ganglion with alcohol, when the needle was placed too far internal, the pain produced by the alcohol was projected backward and into the shoulder, etc., and when placed too far external the pain was referred to the upper jaw—that is, when the side of the ganglion upon which the Vidian enters it was injected the pain was referred posteriorly, whereas when the side upon which the second division entered it the pain was referred to the upper jaw. When the alcohol was placed in the ganglion direct the pain was referred in both directions. (The relative position of the Vidian and second division of the fifth is shown in Fig. 9, which portrays the foramina through which they pass at these points.)

These cases of recent origin (six months to two years approximately) when well of the suppuration were usually well of all symptoms and remained so until a coryza infected the general nasal cavity, when they again developed their pains, often without suppuration of the sphenoidal and postethmoidal sinuses. The membrane of the sphenoidal sinus, however, usually showed under these circumstances a swollen, edematous condition (even polypoid) analagous to that shown by the anterior ethmoidal region in hyperplastic ethmoiditis under similar inflamamtory influences. In the recovery the swelling and edema would subside, but more or less of the pain would continue, and at a time later begin a cyclical reappearance, with no visible disturbance in the sphenoid sinus, even when its anterior wall had a permanent opening in it large enough to permit a good view of its interior. I have observed a similar behavior of the sphenopalatine ganglion, i. e., intermittent reappearance of the neuralgia. I have also observed in these cases a picture identical with the neuralgia which starts in the sphenopalatine ganglion. This is not surprising —on the contrary, would be expected when one proves the accessibility of these nerve trunks. The entire nerve supply of that ganglion—namely, the second division of the fifth and the Vidian nerve—being so closely associated with the sphenoid sinus, the picture of the ganglion neuralgia is readily reproduced under inflammatory conditions within the sinus. The differential diagnosis, it seems to me, is made by the facts —that cocainization of the ganglion stops the pain when it is originated in the ganglion, but fails to stop it when it is originated in the sphenoid, because the ganglion is peripheral to that point of origin; and that cocain solution applied within the sphenoid sinus does stop the pain. In the case of ganglion neuralgia, the membrane covering the sphenopalatine foramen is often, but not always, congested. This appearance is absent in the case where the pain is started in the nerve trunks from within the sinus.

I have seen this class of sphenoidal cases behave as vasomotor rhinitis or rhinorrhea or "hay fever," as the patients call that form of paroxysmal sneezing, accompanied by profuse watery discharge and by asthma. In these cases all therapeutic measures applied peripherally (intranasal) were of no avail. The cases yielded to intrasphenoidal applications. The one so far most usually acceptable has been a 2 per cent to 5 per cent solution of sodium salicylate.

The history of one of these cases bearing on this phase of their behavior I herewith relate:

Case 1.—Miss M. M., thirty-seven years old, consulted me on February 26, 1908, for general headache with great morning sneezing. There was swelling of the cavernous tissues in the nose with profuse general watery secretion; but no pus or visible evidence of sphenoidal inflammation. In the effort to control this syndrome the anterior nasal nerves were injected with alcohol (Stein) and trichloracetic acid applied to the tubercle of the septum (Francis) and middle turbinate, finally the galvanocautery to lower turbinates and tubercle, November 6, 1908; December 26, 1908. The nose was opened for air, but the sneezing continued. All efforts failed. She had to abandon a busy and very useful life for an indefinite period of rest. Dr. D. B. Delavan, who saw her in consultation, advised rest in the mountains. After six months in the mountains of the South she returned to St. Louis, well, and remained so for one year. At this time, March 17, 1910, she returned for treatment because of a "bad cold and headache," as she expressed it. She showed a bilateral postethmoidal sphenoidal suppuration, for which I eventually operated, both sides (April 21, October 27, 1910). On January 10, 1911, she returned to me for an acute coryza with general headache and almost insufferable sneezing (sneezing to exhaustion), without evidences of postethmoidal sphenoidal suppuration, for which she was again compelled to retire to the mountains. June 12, 1911, she again reported to me well. October 12, 1911, she consulted me because of intractable sneezing and general headache with intense burning, stinging sensation in the nose ensuing upon a coryza. This time I began the intrasphenoidal application of 1 per cent cocain in oil, with 1 per cent phenol in oil, which controlled the sneezing and headache fairly well. A little later I began applications of 2 to 5 per cent sodium salicylate water solutions, which proved much more satisfactory—the sneezing and headache stopped in about six days. February 17, 1912, a coryza again brought on general headache and sneezing. This time, knowing that salicylate of methyl was a more potent remedy than the soda salt, and that it was usually tolerated, I filled the sphenoid with a 5 per cent solution synthetic methyl salicylate solution in oil. To my surprise and disappointment, it aggravated the entire syndrome in great severity, and in addition produced asthma lasting five days. A little later, after applications of the sodium salt, she became comfortable; and this in a much shorter time than in the preceding attacks. Locally this case behaves like a hyperplastic ethmoiditis. In the inflammatory stage the membrane becomes edematous with polyp formations, which subside when the attack is over.

That postethmoidal sphenoidal inflammation may give rise to a periodic headache, which is usually spoken of as migrain, is illustrated by the following history:

Case 2.—J. H. A., thirty-seven years old, strong, healthy man, had never had a headache in his life that he remembered; consulted me May 27, 1910, because of a severe left-sided headache which had lasted three weeks. He showed a left sphenoidal postethmoidal suppuration. His pain was parietal and occipital, for the most part, and irregular in severity, sometimes stopping. When severe it was combined with pain in brow and upper jaw. He was melancholic. Treatment was not satisfactory. (He was compelled to be out of the city five days of every week.) On October 12, 1910, he took the proposed operation. The reaction was severe. He was free of symptoms in three months. March 4, 1911, he consulted me because of a coryza with pus which recovered spontaneously. January 6, 1912, he returned because of a coryza with headache, which did not recover spontaneously. At that time he did not show pus in the sphenoid. He had suffered greatly from intermittent general left side headache. Believing that the anesthetic qualities of carbolic acid could be soaked into the nerve trunks in juxtaposition to the sphenoid, I filled the cavity with 1 per cent solution in oil, which stopped the pain in six hours. The cavity was filled once a week for three weeks, when he seemed well, and remained so until the next coryza, six weeks later, which was controlled by 1 per cent carbolic acid in oil. The left pupil is larger than the right during his attacks, and use of his eye is somewhat difficult at those times. He says his pupils were always equal before his headache began and are so now in the intervals between coryzas when he is well of headache. This case has so far not behaved as a hyperplastic ethmoiditis. In the inflammatory attack it shows only redness with little swelling, and sometimes pus.

Case 3.—Mrs. Buc., aged forty years, consulted the Nose and Throat Clinic, O'Fallon Dispensary, Washington University Medical Department, for severe headache, worse on left side, extending to neck and shoulders. All examination was negative. After considerable effort I determined a postethmoidal sphenoidal inflammation right and left, and later, February-July, 1910, opened both sides. The reaction was severe. After some months she was better, and finally became comfortable. A coryza without suppuration later reestablished the pain, which on the right side was stopped by intrasphenoidal applications of 10 per cent salicylate of methyl, and much improved on the left side. Applications of cocain inside the left sinus quickly produced total anesthesia and analgesia of the areas supplied by all the divisions of the fifth nerve, with a marked sense of stiffness of the lower jaw of that side, which I took to be the effect of the cocain upon the motor function in the third division. Dr. D. E. Jackson, of the Pharmacological Department, Washington University, agrees with me in this conclusion. The right side ceased to be painful six months ago, which is shortly after the applications of methyl salicylate were begun. The left continues to be intermittently painful to a considerable degree, but is markedly helped by methyl salicylate, and remains vastly better than before this treatment was begun. She frequently complains of stiffness of the lower jaw on that side. Only once did this case show a tendency toward edma with polyp formation at the time of acute inflammatory attack, and then only slight.

Case 4.—Miss A. McM., thirty-eight years old, consulted me March 20, 1911, because of intermittent headache from which she had suffered for years. At this time it had been coming much oftener and has been rapidly growing more severe, then requiring 2½ grains of morphin sulphate subcutaneously administered by her physician to control the pain. She never took morphin herself. The pain was worse in left temple; but in the height of the attack it seemed to extend over the entire head. And very rarely it came on the right side alone or would be much worse on that side. A postethmoidal sphenoidal inflammation was found on the left side, which was operated two months later. The reaction was intense. She improved little or none during the next four months under a treatment of simple cleansing of the cavities. At this time (October, 1911), intrasphenoidal applications of 10 per cent methyl salicylate were begun with the idea of soaking the medicament into the environing fifth nerve. Since then the general improvement in her suffering has been very considerable. Much of the time she is free of pain, and a large part of the remaining time she suffers only slightly. The recurrence of the intensely severe attacks is less frequent and intense. She recognizes two kinds of pain—one that I can stop by filling the sphenoid sinus with cocain methyl salicyate or menthol solutions, and another distributed over the same area which I have tried in vain to stop by these measures. When this attack begins it progresses despite all efforts to stop it; but it has become less severe and less frequent under this treatment, and the last attack, April 1, 1912, was on the right side only.

This case has never in any way suggested by its clinical behavior a similar condition to that of hyperplastic ethmoiditis. It has always appeared a simple inflammatory condition of low grade with once a suppurative stage also of low grade. Since the primary appearance disappeared this district of the nose has appeared normal.

Case 5.—Mrs. C. Siek, aged twenty-five years, consulted me February 20, 1903, for an acute pansinus suppuration with general headache. Prior to this attack she had very rarely in her life been sick in any way. She did not remember to have ever had a bad headache, and had always been strong. She has done hard work (shoe machine) since her nineteenth year. Since this attack she has had, up to the present time (nine years), a recurrent headache that seldom skips more than ten days. Much of the time it is frontal, sometimes maxillary, and often intense combined with occipital pain. It is worse at night and during menstruation. Meanwhile I have opened the frontal sinuses and antra and ethmoids and sphenoids, and done everything known to possibly help (contacts, etc.). I have from time to time put her in charge of specialists in all the departments of medicine, who have found her normal, save hyperopic astigmatism, for which she wears glasses. November, 1911, I proved that her trigeminus was easily accessible (intrasphenoidal) on both sides. Since then I have filled the sphenoid once in ten days with 1 per cent phenol in oil. She says that this is the first medication to help her pain.

All characteristics of a nose case disappeared from this patient by the end of the first three months.

I could report another case similar to this, of four years' standing, in which there has been a recurrent sphenoidal suppurative inflammation at long intervals with great exacerbation at these times.

Case 6.—Mrs. M. D. A., forty-eight years old, consulted me

June 11, 1910, because of right side recurrent sphenopalatine ganglion neuralgia, or, at least, what seemed to be such. It had lasted twenty-four years. It was stopped by cocainization of the sphenopalatine ganglion and controlled for one year by carbolization[2] of the ganglion, at the end of which time she suffered some kind of an explosion which took in her entire head, upper and lower part, on both sides, which confined her to her bed for four weeks despite acetphenetidin, aspirin, codein and morphin. I did not see her during this illness. After this attack, however, she came to see me, but showed no local changes. She continued to suffer very greatly and frequently from what seemed, from its distribution, a trigeminal neuralgia with pain in her occiput and neck, almost altogether on the right side. After a careful explanation of her case, describing the local conditions and telling of a possible benefit from intrasphenoidal medication, Dr. Bliss and I advised her to submit to the opening of her sphenoid, despite the fact that no sphenoidal inflammation betrayed itself. She accepted the proposal. Twice prior to the Hajek radical postethmoidal sphenoidal operation I injected a drop of 5 per cent solution carbolic acid[2] in 95 per cent ethyl alcohol into the sphenopalatine ganglion, each being allowed to rest five days with the idea of benumbing it and thereby reducing the reactionary shock. The pain of the operation under cocain and the succeeding depression and pain were, in this case, astoundingly little. Subsequent filling of the sphenoid sinus with 2 per cent sodium salicylate water solution has been accompanied by a decided betterment, both in severity and frequency of the pain on the right side.

Case 7.—Miss M. R., twenty-one years old, consulted me March 24, 1912, because of a mild posterior headache with dilatation of the pupil on the right side, accompanied by discomfort in use of her eyes, of one week's standing. Ophthalmologic examination was negative. An upright rod appeared to her bifurcate below, which, I understand, indicates a paresis of the superior oblique. The anterior wall of the right half of the sphenoid was reddened and bathed in serum. The effort was made to fill the sinus with 1½ per cent sodium salicylate water solution. This was repeated three times in nine days, when she was free of all signs and symptoms, and remained so ten days, when she had a "sick headache" (in which I did not see her) and again had a dilated pupil which became normal when the headache stopped. No discernible cause could be assigned for the sick headache.

The difficulties that beset some of these diagnoses need not be considered by me. They were emphasized by Dr. Casselberry in his paper before this association last year. I have selected a series of cases which pass by graded stages from the most evident to the least evident, if indeed Case 6 be admitted to the company of sphenoidal cases. These cases are not unique. They have been selected from a considerable number. Many times have I seen the same features in other cases, with the exception of Mrs. M. D. A., Case 6, in whom I opened the sphenoid, which appeared normal, in order to permit of intrasphenoidal medication in the effort to soak the remedies through bony walls directly into the nerve trunks. It has been of apparent benefit under extreme circumstances; but the result is not of sufficient time standing to admit of conclusions. In the effort to forestall the shock and subsequent reaction and pain of Hajek's radical postethmoidal sphenoidal operation I have five times carbolized the sphenopalatine ganglion with marked success.

Some of the other cases have had optic atrophy. The vision would diminish under coryzas. Pain lasted much of the time.

In the observation of these cases I am led to the belief that a large number of the frequently recurring headaches, of whatever length of time standing, that usually bear the name "migrain," that are met with in the general practice of medicine, that have defied diagnosis and treatment, are sphenoidal inflammations existent, or were started as such. They may have lost all the evidences of local disease, which some months before were easily recognizable. I believe that the nerve trunks have become diseased from juxtaposition, just as the optic is known to do. Disease of the nerve trunks under these circumstances is in nowise different in its clinical behavior from that which is started further toward the periphery, as, for instance, from an antrum of Highmore suppuration or a diseased tooth, which after the cure of the local disorder continues painful at intervals or becomes a tic-douloureux. The second division of the fifth in the foramen rotundum is in almost as close association with the sphenoid sinus as is the optic nerve in the optic canal, and under identical setting, namely, surrounded completely by a firm, bony ring. This is true of the third division of the fifth also; but it is not, as a rule, so closely associated with the sphenoid sinus. Optic nerve disease, secondary to the sinus disease, is too well known and proven to require argument from me. I see no reason why we may not assume as much for the other nerve trunks, in close association with these sinuses, with the difference between motor and sensory nerves.

This sphenoidal area being central to the place of injection of alcohol into the branches of the fifth nerve, from under the zygoma, may explain some of the cases that have not been relieved by that treatment. Also, for the same reason can we understand that when the Gasserian ganglion proper has become involved and gives rise to the pain, intrasphenoidal applications will fail to relieve.

The involvement of the nerve trunks in the sphenoidal district producing the sneezing cited in Case 1 explains why the injection of the branches in the neighborhood of the sphenopalatine foramen (Stein), combined with the same treatment of the anterior nerves, has failed to influence the sneezing in some cases, the sphenoidal area being central to the other nerves.

It would appear from Case 2 that whatever may be the lesion of the nerves it is rather easily remediable in its earlier stages by intrasphenoidal medication. How far such patients as cases 5 and 6 may be helped by intrasphenoidal medication remains to be learned.

I have not thus far been able to secure any postmortem material bearing on the above question.

The various medicaments mentioned above have been distinctly advantageous in the treatment of some hitherto irremediable cases (at least in my hands). There is, however, much to be desired—many remedies remain untried. (In many respects this paper must be considered a preliminary report.)

That the cavernous sinuses may become infected and thrombotic, secondary to sphenoidal infection, is well known. In these considerations the cavernous sinus, however, plays only an anatomic part.

I have cited in the records of some of these cases that they behaved as a hyperplastic ethmoiditis, and that some did not. So far as I have been able to learn, there is no postmortem histologic proof of a hyperplastic process in the sphenoid such as has been proven in the ethmoid. This possible condition has been thought to exist and reasoned out by Hajek[3] in his third edition. He also laments the absence of postmortem material.

REFERENCES.

1. Sluder, Greenfield: The Syndrome of Sphenopalatine Ganglion Neurosis. American Journal of the Medical Sciences, December, 1910; also Transactions American Laryngological Association. 1910.
2. Sluder, Greenfield: A Phenol (Carbolic Acid) Injection Treatment for Sphenopalatine Ganglion Neuralgia. Journal of the American Medical Association, December 30, 1911, Vol. LVII., p. 2137.
3. Hajek: Pathologie und Therapie der entzuendlichen Erkrankungen der Nebenhoelen der Nase. Wien (3rd ed.), 1908.

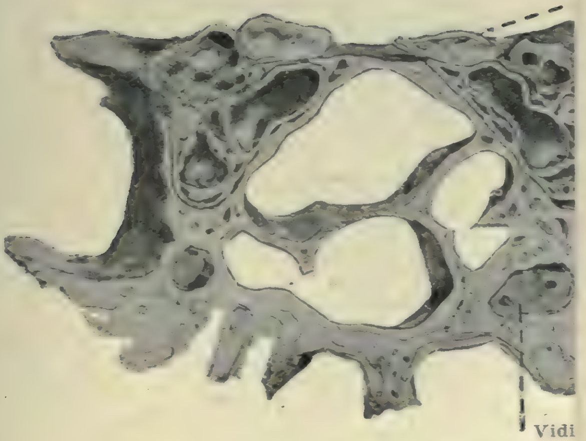


Fig. 1.* (Two diameters.)

Cross section of a cavernous sinus of large lengt
at a point far forward, as indicated by the section th
nerves and anterior clinoid processes seen from behi
nerve trunks well separated from the sphenoidal sinu
shows only the right sphenoidal sinus.

---

*This figure was published in the Archiv fuer L
Band, 3 Heft . An error occurred in this publication
drawing, in the numbering of the nerve trunks. The
3rd, the 3rd was marked 1st div. of 5th, the 1st divi
was marked 6th, the 6th was marked 2nd division of
were all marked "in the optic canal," which, of c
possible.

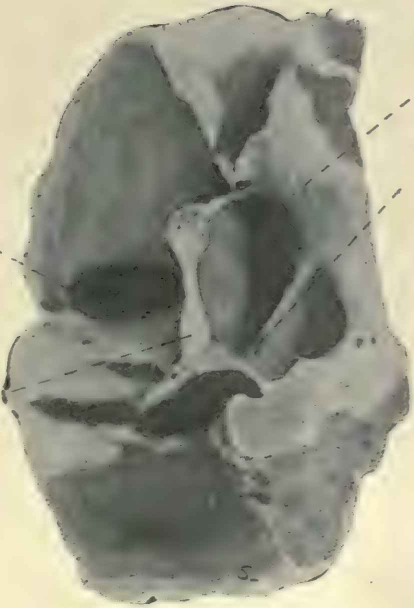

Sphenoidal sinus.
Foramen ovale.
Vidian canal deficient at point of indicator.
Bony wall separating foramen ovale from sphenoidal sinus.
S.

Fig. 2ii. (Two diameters.)

The same specimen as shown in Fig. 2i. Seen from a point above and to the right.

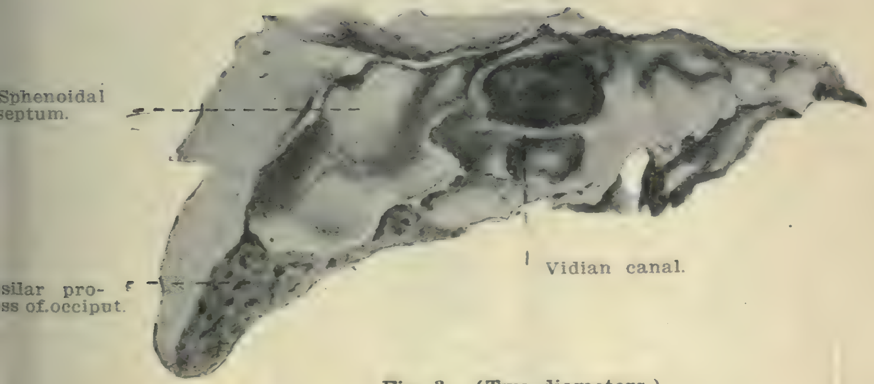


Fig. 3. (Two diameters.)

Interior of left sphenoidal sinus, showing Vidian canal supported by thin bone with the sinus cavity and below it. The canal is open on top.

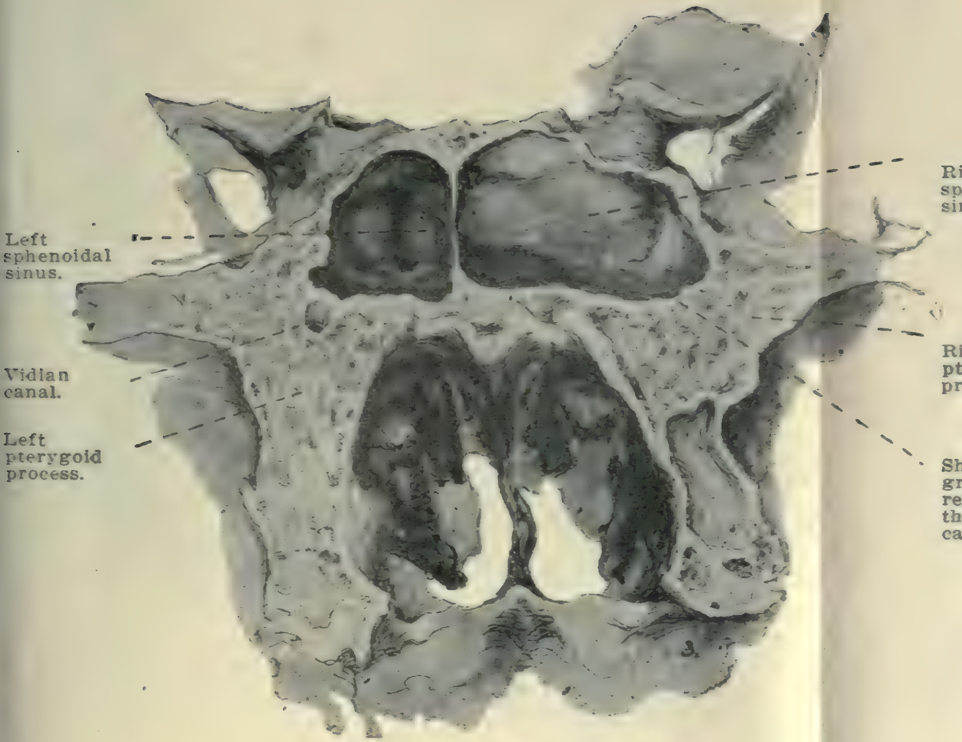


Fig. 4. (Two diameters.)

Cross section of a skull through the pterygoid processes. Seen from behind, showing the Vidian canal of left side well imbedded in the bone; and that of the right side exposed in the sphenoidal sinus.

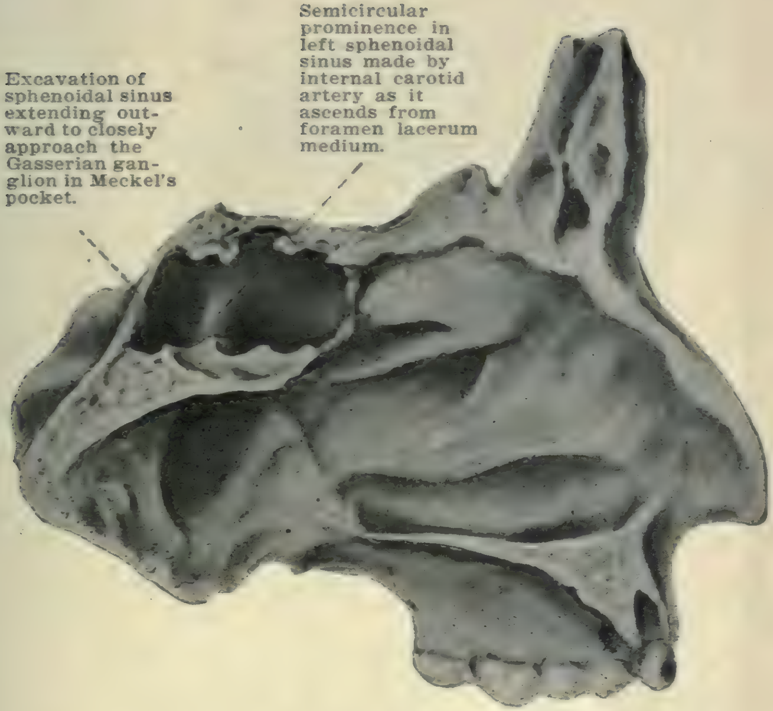

Excavation of sphenoidal sinus extending outward to closely approach the Gasserian ganglion in Meckel's pocket.
Semicircular prominence in left sphenoidal sinus made by internal carotid artery as it ascends from foramen lacerum medium.

Fig. 4ii.

Picture of specimen loaned me by Dr. H. P. Mosher.

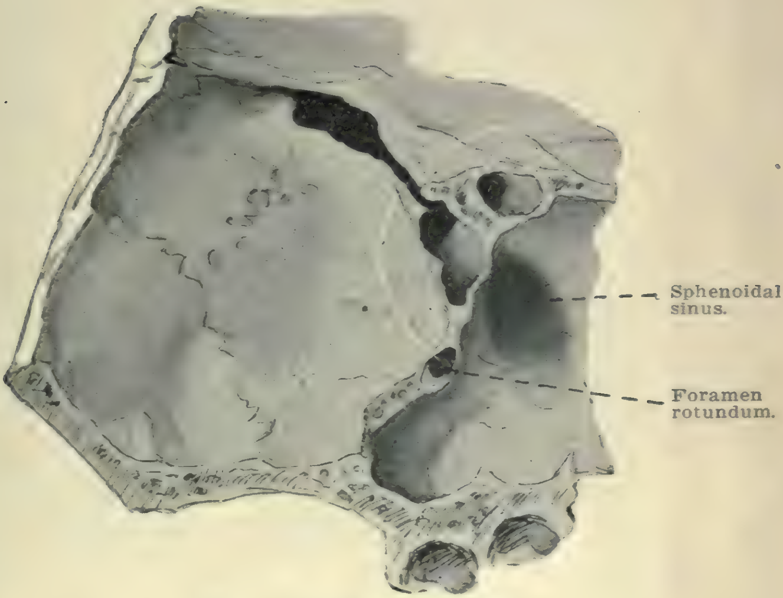


Fig. 5. (Two diameters.)

Middle fossa of skull, showing foramen rotundum separated from sphenoidal sinus by thin film of bone.

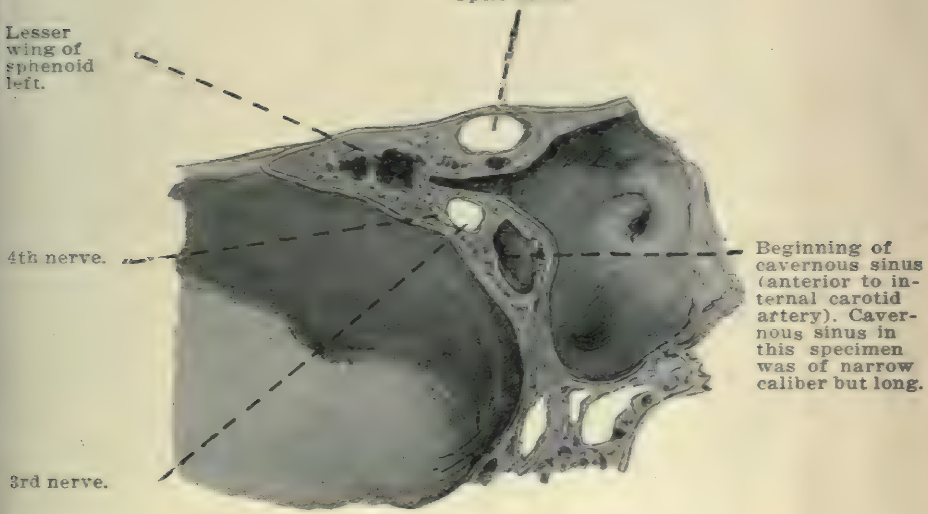

Optic nerve.
Lesser wing of sphenoid left.
4th nerve.
Beginning of cavernous sinus (anterior to internal carotid artery). Cavernous sinus in this specimen was of narrow caliber but long.
3rd nerve.

Fig. 6. (Two diameters.)

Left sphenoidal sinus seen from behind, showing a prolongation into lesser wing of sphenoid, bringing 3rd nerve in close association with it.

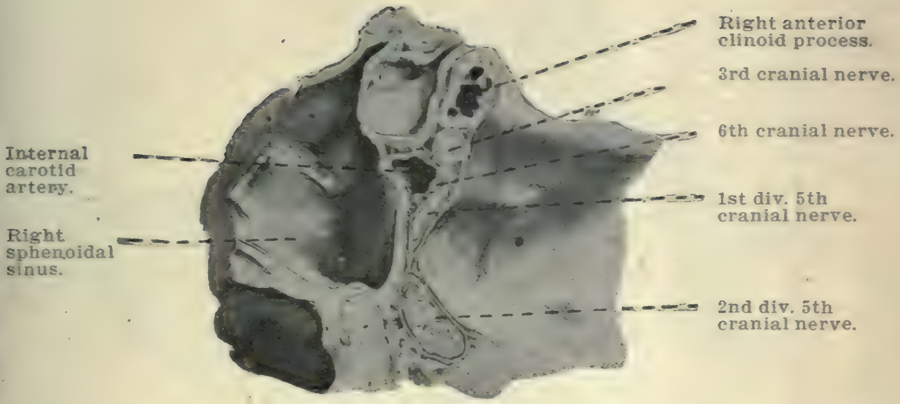

Right anterior clinoid process.
3rd cranial nerve.
6th cranial nerve.
Internal carotid artery.
1st div. 5th cranial nerve.
Right sphenoidal sinus.
2nd div. 5th cranial nerve.

Fig. 7. (Two diameters.)

Cross section of right cavernous sinus (long and narrow), seen from behind, showing first division of 5th nerve in close association with sphenoidal sinus.

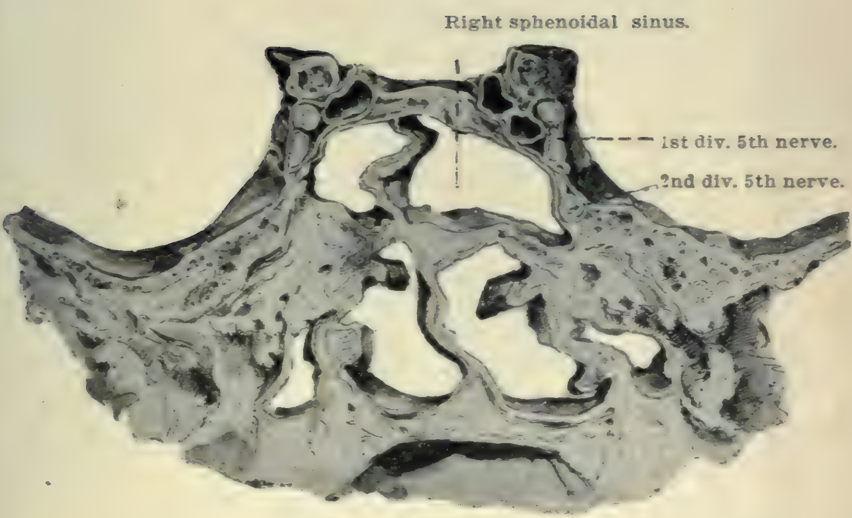


Fig. 8. (Two diameters.)

Cross section of sphenoidal sinus and anterior clinoid processes, showing first and second divisions of 5th nerve separated from sphenoidal sinus by thin bone.

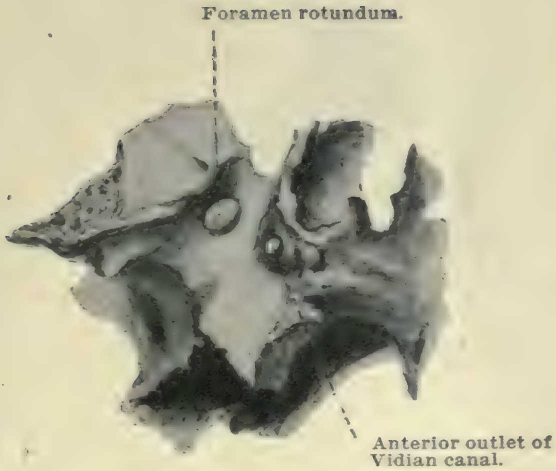
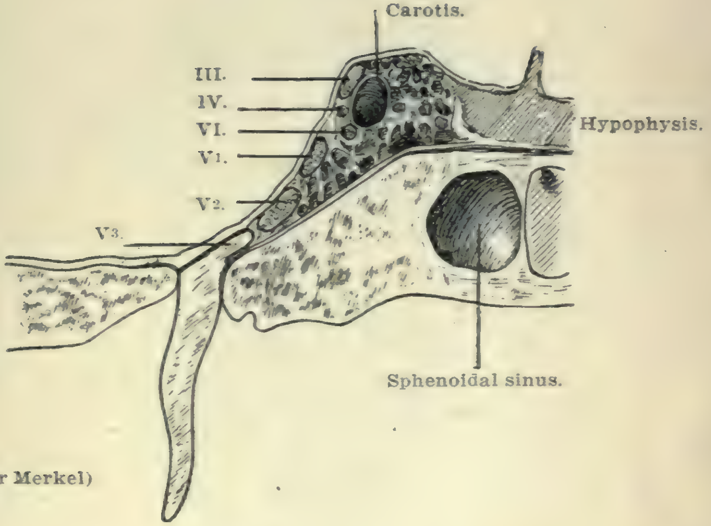


Fig. 9. (Two diameters.)

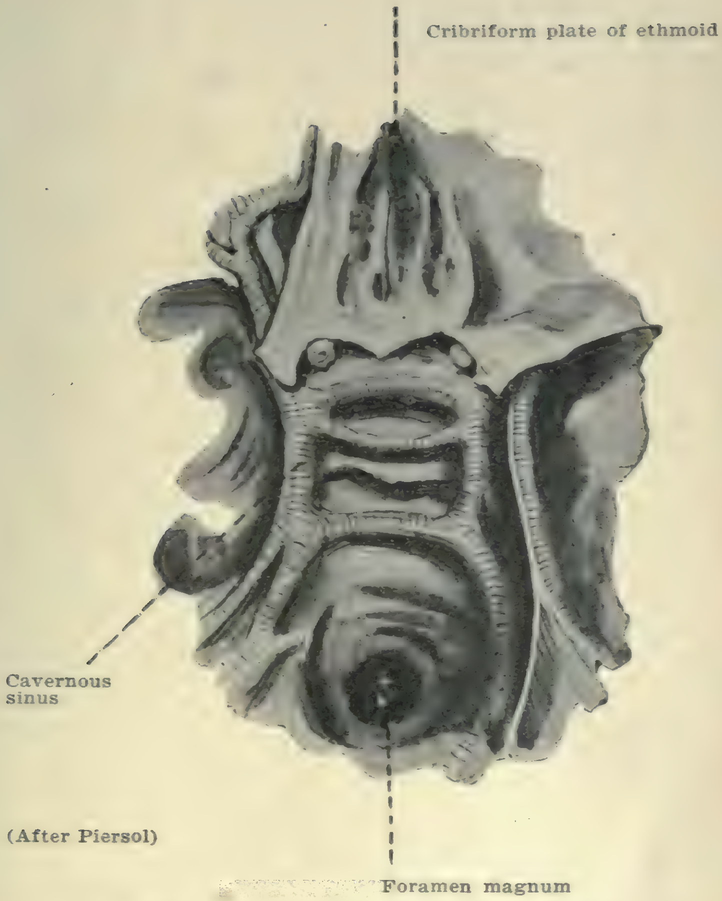
Anterior aspect of base of right pterygoid process, seen from in front.



(After Merkel)

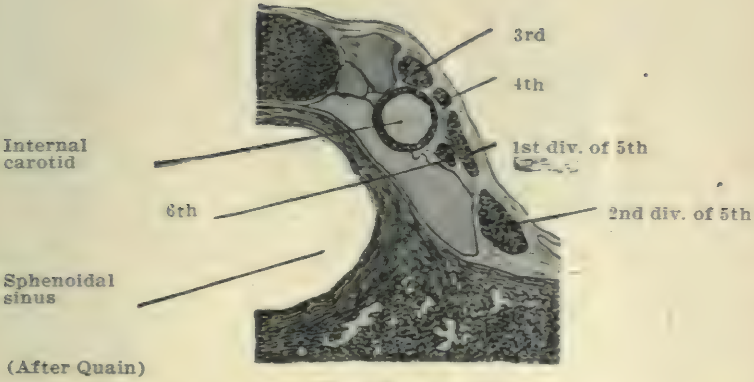
(Two diameters.)

Showing cavernous sinus and third, fourth and first, second and third divisions of the 5th and 6th nerves, hypophysis, sphenoidal sinus, and carotid artery.



(After Piersol)

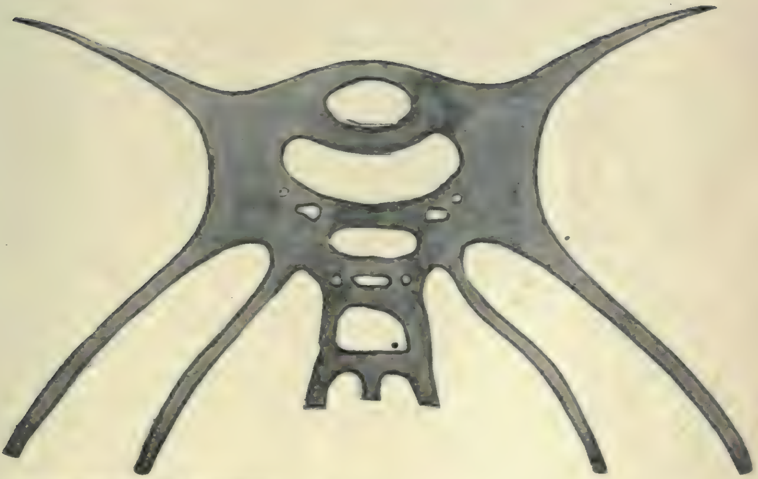
Showing venous formation of cavernous sinus.



(After Quain)

(Two diameters.)

Showing cavernous sinus and third, fourth and first and second divisions of the 5th and 6th nerves, hypophysis, sphenoidal sinus, and carotid artery.



(After Rauber and Kopsch)

Showing venous formation of cavernous sinus, seen from above.

XLIX.

EXHIBITION OF OTITIC BRAIN ABSCESS CASES AND A MASTOID EXENTERATION BY NATURE.*

By S. MacCuen Smith, M. D.,

Philadelphia.

Two of the cases that I have the pleasure of presenting to you today have been shown before the Section on Otology and Laryngology of the College of Physicians of Philadelphia, but are of sufficient interest, I feel, to be again brought to your notice. The first case, however, that of the eight-year-old child, is still under care and shows the results of an evacuated brain abscess formation, especially in the relief of the neurologic symptoms.

In the majority of my cases, I am impressed with the absence of diagnostic data that will enable us to state that an abscess formation is present, or, more particularly, its actual location, with these exceptions: The presence of word aphasia in a left-sided temporal abscess formation, and that indescribable clinical picture as seen in the facial expression. Indeed, I have been compelled in most instances to rely principally on these two symptoms, and regret that I am unable to definitely define the facial expression above referred to, but am sure you have all had a like experience.

Another symptom, which has been so constant in patients coming under my observation that I am inclined to believe it pathognomonic of an abscess formation, involving the temporosphenoidal lobe of the brain, is the very severe pain in the frontal region of the affected side. Indeed, this symptom has been so unfailingly present and so severe in the average case, that one might readily be deceived in thinking the abscess formation was in the frontal lobe.

---

*Read before the Eastern Section of the American Otological, Rhinological and Laryngological Society, at Philadelphia, February 22, 1913.

Of course, in the small minority of cases we are able, by certain tests, including the eye, blood and neurologic examinations, to state with more or less accuracy that a given patient is suffering from a brain abscess formation, and we might even be able to state its location with some degree of certainty; but if these same rules are applied to the average case, we would fall far short of proving our point. So, after all, personal observation, together with intuition, which comes only from large experience, must be depended upon to aid us principally in our diagnosis and localization of intracranial lesions complicating aural disease.

I wish again to bring out one point which I feel is of the greatest importance: When possible, follow the path of infection through the tegmen antri or tegmen tympani, when searching for a brain abscess formation, as this not only favors drainage by gravitation, but also lessens the possibility of lighting up new infection by going through the skull at some other point.

Case 1.—D. P., aged eight years, was admitted to the Jefferson Hospital on October 24, 1912. Her family history was negative. Three years previous to this time the child had scarlet fever, complicated by an acute otitis media of the left ear, since which time the discharge has been constant except for two periods of about three months each, when it ceased. Just prior to the acute exacerbation the child would become especially irritable and mentally confused.

In the spring the child became restless and quarrelsome, lapsing from her naturally cheerful disposition to one irritable and morose, and growing progressively worse. About a month before she appeared for treatment, she fell and struck on her head, in the frontal region. Following this blow she had severe, persistent frontal and left temporal and parietal headache, with extreme nervous irritability, vomiting and intermittent delirium, principally nocturnal, and was able to retain but very little food. One week after the injury she had a convulsion.

Examination on Admission.—The child is fairly well nourished, temperature normal and pulse 53. Chest and abdominal examinations are negative, as are also the laboratory reports on blood and urine, and the neurologic and ophthalmologic reports.

Left Ear.—The canal is filled with a quantity of brownish-yellow, foul-smelling pus. There is moderate drooping of the superior and posterior wall of the canal, but no tenderness over the mastoid process. There is entire absence of the tympanic membrane and ossicles, and the mucosa lining the tympanic cavity is very red. The X-ray report, by Dr. Manges, shows the mastoid cells on the left side filled with pus. The right side is normal. Skull has very much the appearance of an internal hydrocephalus, but there is no evidence of brain abscess.

Operation.—A radical mastoid operation was performed on October 29th, or five days after admission. The cortex of the mastoid was very hard, and upon its removal the antrum was found to be almost obliterated. The sinus was in about a normal position. There was a carious exposure of the dura through the tegmen antri about one-fourth of an inch in diameter. The walls of the antrum and the few cells that still existed were covered with a peculiar membranous material.

The case followed an uneventful course of convalescence after the mastoid operation, but the marked irritability continued.

A Wassermann test was positive and the patient was placed on inunctions and potassium iodid, without any change in the clinical picture.

The child was allowed to return to her home in central Pennsylvania in about one week, owing to her extreme homesickness, but on the following day she had a second convulsion, and was immediately brought back to the Jefferson Hospital, where she was kept under observation for two weeks, when she developed measles and was sent to the Municipal Hospital, whence she returned to her home one month after the mastoid operation.

For a fortnight her condition was much improved, when suddenly, without apparent cause, the severe headache over the left frontal region returned and her mental state became noticeably worse. She could not recall the names of people whom she had known all her life, nor associate names with common objects. For instance, she called a knife a ring, but said it was used to cut. Her chief complaint was headache.

The patient was returned to Philadelphia and admitted to the Germantown Hospital six weeks after the mastoid operation. An examination showed the ear to be in good condition. The temperature was 98.8°, the pulse 98 and respiration 26. The urine and blood examinations were negative.

The eye report showed pupils equal, reaction normal except outward rotation of the right eye, which was practically nil. Eye grounds, media clear; discs swollen and margin everywhere obscured; best seen with a + 5. Retinæ edematous, blurring the blood vessels. Small veins tortuous. No hemorrhages seen. (Choked disc.)

The neurologic report showed no exaggeration of patellar reflexes. No ankle clonus. Babinski slight on both sides. Romberg's sign present on both sides, worse on right. Some rigidity of muscles at nape of neck. Masseter muscles contract firmly on both sides. There seemed to be no paralysis involving facial nerve on either side. Paralysis of abducens on each side. Grips equal, but not vigorous on either side. Patient was extremely irritable and suffered from almost constant frontal headache.

Four days after admission to the hospital, under general anesthesia, we reopened the mastoid wound, with a view of exploring the interior of the skull, hoping to locate and evacuate the suspected brain abscess. The process of repair was found to have gone on very rapidly. The exposure of the dura was enlarged by the removal of a lateral plate of bone for a distance of about one-half inch. There was no pulsation discernible in the dura and it seemed to be under considerable pressure. The dura was incised and a pair of Jackson forceps was introduced towards the temporosphenoidal lobe, along the avenue of invasion, for a distance of about one and one-half inches, when there was a free escape of a greenish, odorless pus, two ounces of which were collected in test tubes and it was estimated that about another ounce escaped. A drain was introduced, and the patient returned to her bed in a decidedly exhausted condition, it being necessary to employ normal salt solution intravenously, after which she reacted well. An examination of the pus showed few organisms present, staphylococcus pyogenes albus predominating. No tubercle bacilli.

Twelve days after the operation the patient was totally relieved of all suffering, her mental state was virtually normal, and she could recall many incidents that occurred several years previously.

Six weeks after the brain abscess operation, notwithstanding our efforts to keep the external wound open, it had closed sufficiently to require reopening, which was done under general anesthesia.

Case 2.—W. S., aged thirty-seven years. When about twelve years of age he had an attack of scarlet fever, complicated by a suppurative otitis media of the left ear, which had been recurrent ever since, or for about twenty-five years. The longest period between attacks was from two to three years. During this time he maintained a state of good health. On two different occasions, about ten years ago, a polypoid mass was removed from the ear.

Examination showed the left ear well filled with a brownish-yellow, foul-smelling discharge, escaping through a large perforation in the posterosuperior quadrant of the drumhead, involving almost one-half of this part of the membrane. A large mass of granulation tissue practically filled the tympanic cavity. Considerable discharge was also escaping through a large carious opening about the middle of the posterior osseous canal, through which a probe was passed directly into the antrum, revealing much carious bone. The patient suffered from attacks of vertigo about once in each week, of sufficient severity to cause him to fall unless he promptly obtained some support,

The patient believed that he was entirely deaf in this ear, although he could hear the tuning-fork in close proximity to the concha. Bone conduction was greatly reduced from normal. No tenderness was elicited on pressure over the mastoid.

Because of his apparent good health and the absence of suspicion of intracranial lesions, the usual preliminary examinations of blood and eye grounds were not made.

Operation, March 4, 1909.—A radical mastoid operation was performed at the Jefferson Hospital. The cortex was found to be very thick and hard, with the exception of the mastoid tip, which was soft and necrotic. The antrum was reached with difficulty, on account of the dense, eburnated bone. As soon as the antrum was opened a quantity of pus gushed out, as though it had been under some pressure. When this was cleaned away the pus was seen to escape through a carious opening in the tegmen antri and through the dura. A probe was introduced through this opening into the interior of the skull, the sinus apparently leading into the temporal lobe. A pair of Jackson forceps was then introduced and separated, permitting the escape of additional pus. Proper drainage was provided and the patient made a slow but uninterrupted recovery.

The chief point of interest in this case is that the patient apparently suffered no inconvenience at any time from this pus formation in the interior of the skull, unless we may attribute his spells of vertigo to this cause. It would, of course, have been most interesting to have had a record of his visual fields and a differential blood count.

The ear at this time, four years after the operation, looks perfectly well in every respect. Complete dermatization took place within one year, and the patient now hears the tuning fork at about two and one-half inches.

Case 3.—E. C. B., aged twenty-five years. Car inspector. Patient had measles when two or three years of age, and complicating this disease an acute suppurative otitis media of the left ear. The discharge in this ear continued recurrently up to one year ago, when the patient applied for treatment at the Jefferson Hospital, and just at that time was especially active and profuse. For about a month he had suffered from vertigo, which was increased by bending the body or by quickly turning the head, in any direction.

When first examined the external auditory canal was well filled with a collection of pus and desquamated epithelium. Upon the removal of this mass we found an entire absence of the membrana tympani and ossicles, they having become disintegrated and escaping in the discharge probably some years ago. A bent probe introduced into the antrum shows that the cells comprising the tip have become obliterated through sclerosis. The greater part of the superior and posterior osseous canal is destroyed, from the tympanic cavity back to the mastoid antrum, all of which, except the tympanic end of the eustachian tube, seems thoroughly dry and shows complete dermatization.

Bone conduction is little, if any, reduced. Hearing for C tuning fork, aerial conduction, is only one-half inch.

Various tests indicate that the patient has a marked vestibular disturbance, more especially involving the superior and horizontal semicircular canals. The whole picture shows in an unusual manner nature's efforts to provide for evacuation of the inflammatory débris by performing essentially a radical mastoid operation.

REPORTS.

X-Ray.—Left mastoid is of a chronic sclerotic type, with considerable bone defect in the region of the antrum cells. The lateral sinus is not superficial and does not appear to be far forward. The mastoid process on the right side is apparently normal. (Dr. Manges).

Neurologic.—We find no neurologic symptoms in this patient other than deafness and vertigo. There are no central symptoms. He is not hysteric nor neurasthenic. (Dr. Price.)

Ophthalmologic.—Patient is suffering from compound myopia and requires reglassing. Otherwise the eyes are healthy. (Dr. Le Fevre.)

L.

A REPORT OF THREE UNUSUAL CASES OF STAMMERING.*

By G. Hudson Makuen, M. D.,

Philadelphia.

Stammering is like many similar nervous affections or diseases in that it may be divided into three more or less distinct stages: the prodromal, the acute, and the chronic. The prodromal stage is found in early childhood, and it is characterized by rapid and blustering speech. The acute stage comes in later childhood, and it is characterized by a marked hesitation in speech, which is usually unconscious, although accompanied by varying degrees of spasmodic muscular contractions and contortions. The chronic stage follows hard upon the other two as the natural result of a continuance of the affection, and it has all the symptoms of the other two in a somewhat aggravated form, together with certain important mental and emotional complications which always accompany a full, conscious realization of the condition.

The prodromal stage is one from which many children pass without serious difficulty, especially when they have some intelligent help; the acute stage is the forerunner of more serious trouble, and the chronic stage is the full development of the affection with all its varied complications.

Every case of stammering is unusual because no two are exactly alike, but some cases are more unusual than others. The three which I am about to report are alike in that they are all in the chronic stage, two of the men afflicted being forty years of age and having stammered for more than thirty-five years, and one of them being thirty-two years of age and having stammered ever since he can remember.

Case 1.—E. K., a physician, forty years of age, has stammered for thirty-five years. His stammering varies in degree and it has increased in intensity. There is no history of hereditary influences, nor was the patient associated as a child with any similarly afflicted. He had whooping cough at an early age, but it was not sufficiently severe to be a factor in his condition.

---

*Read before the Eastern Section of the American Laryngological, Rhinological and Otological Society, February 22, 1913.

The patient is in good health and possessed of all his faculties. He is nervous and excitable, although he claims that this condition is a result rather than a cause of his stammering. He is of average mentality, has a good memory, is strongly initiative, and does not appear to be lacking in attention, although his powers of concentration and volition are unstable. His family history reveals nothing of a positive character except that his mother died of tuberculosis. The patient himself is unable to recall any particular incident in his life that may suggest a cause for the beginning of his speech difficulties. There is no peculiarity about his writing. He stammers somewhat when he attempts to read aloud, he stammers very much in conversation, and he is quite unable to speak at all in public. The defect does not appear in singing.

He claims to have had no subjective nose, throat or ear trouble, although the septum is markedly deflected toward the left nostril, causing considerable intranasal pressure and pharyngeal irritability; a condition, by the way, which I have found to be very common with stammerers. More than 90 per cent of my cases have intranasal pressure with irritability of the pharynx.

The patient had taken a course of treatment in Philadelphia and had been twice to a Detroit school for stammerers, but he had received no real benefit whatsoever. His conversational speech, when he was able to say any words at all, was drawling and painful to listen to, and it was accompanied by various motions of the hand and forearm, such as beating time and drawing figures of eight, which maneuvers he claimed made it possible at times for him to surmount his difficulties to some extent, and occasionally at least to converse with his friends because they enabled him to forget his stammering and thus to banish the fear of it.

His own experience had led him to the belief that stammering is a "phobia," pure and simple, and that there is no help for the affection except through the removal of fear. He was convinced also that all training of the organs of speech must be worse than useless, because it tends to fix the attention upon the difficulties and thus to increase the fear. He was sure that he could speak well if he could banish fear, but he was equally sure that there was no power on earth that could help him to do it.

He was in Philadelphia about two weeks, during which time I saw him occasionally and tried to convince him of the error of his ways and the falsity of his theories, but I failed utterly because he was so disinclined to do the amount of work which would have been necessary in his case to enable him to acquire volitional control of his organs of speech, and thus prove to himself that there was no basis for his fear. It was of no avail that I quoted Emerson where he says, "The antidote to fear is knowledge." The fear of stammering always vanishes when the patient has acquired a good working knowledge of the mechanisms of speech and a voluntary control of them through systematic and persistent practice.

This man, however, was in the unfortunate condition of mind, which is by no means uncommon in these cases, of being unable to make any persistent effort to do what seemed to him a great amount of work, because of his doubt or lack of confidence in the results to be acquired. And who can blame him? He had had three unfortunate experiences with other methods for a cure. He had given his money, his time, and a certain amount of energy to the work without receiving an adequate return, and his confidence was not only shaken but absolutely shattered. I could not persuade him to try again. It was in vain that I pointed out to him the difference between the methods which he had used and the one which I was advocating. I tried to show him that the former were merely tricks to divert his mind from the difficulties and thus enable him to overcome them, while the latter system, or the one which I would have him adopt, was along physiologic lines and consisted merely in an effort to supplant his defective speech with normal speech, built up on scientific principles. I was utterly unable to convince him that the end would justify the means, and he went away somewhat in the condition of the man who said that "it was happier to be miserable than to be happy without being miserable."

Case 2.—Mr. R., also forty years of age, has stammered for thirty-six years. His stammering is not increasing because, as he said, it cannot be worse. It was of gradual development and it began spontaneously. When a child, he says, he was "saturated with the Bible," and he was once greatly disturbed by overhearing a conversation in which it was predicted that the world was soon coming to an end. He gives no history of relatives having stammered in their speech, but he says that his mother, who was also sensitive on the end of the world question, had something akin to stammering in her fingers while playing the piano. He has one younger sister, who has no defective speech whatsoever. He had whooping cough and measles during infancy, and he suffered from some intestinal colic when very young. His memory is fairly good, but his powers of concentration are poor.

The patient's father developed epileptiform attacks late in life, which interfered with his speaking and writing during the last two years of his life.

The patient is exceedingly nervous and excitable, a condition which he thinks he inherits and which is largely responsible for his trouble. In 1884 he studied some months with a negro who had himself been cured, and thus he got some temporary relief. In 1890 he was in a school here in Philadelphia for the cure of stammering, and again in 1894. In November of 1911, he consulted Dr. Coriat of Boston, who attempted some mental or psychic method of treatment, but he was unable to put him in the hypnotic state, or to find any subconscious cause for his condition. The theory is now being advanced by certain neurologists or psychologists that stammering is due to a "forgotten reminiscence," such, for instance, as this man's fright of early childhood with reference to the ending of the world, and that this forgotten reminiscence must be recalled or brought within the domain of the consciousness before the stammerer can be permanently cured. Professor Munsterburg of Harvard was nearer the truth in the matter, however, when he said he had long since ceased to experiment with stammering, because the psychic method halts when it reaches the physical obstruction.

Case 3.—G. G., aged thirty-two years, employed in a wholesale drug house, had stammered ever since he could remember. His father was an alcoholic and died in an insane asylum.

When he was a baby, his father came home intoxicated one evening and upset the cradle, greatly frightening the child and every one in the house.

The patient had none of the infectious diseases of childhood, and there was no one else in the family who had been similarly afflicted. He was mentally alert and fairly bright, but inclined to be introspective, moody, and at times even morose. He seemed to brood over his condition, but not, as I thought, excessively or more than he could bear, and he began his treatment with us in a fairly satisfactory manner, making very good progress, indeed. His sister called upon us after two weeks had elapsed and expressed herself as greatly pleased. She said that he talked better and he was much less nervous. After about five weeks, however, he made a little visit home, and his uncle showed some disappointment and displeasure, because as he said, he seemed not to be entirely cured.

This greatly discouraged the patient, and he came back to us in a very nervous and excited condition. My assistant, at whose home he was staying, said that she was anxious about him because he seemed at times not to be able to control himself or to do his work satisfactorily. He would draw his hand across his forehead and stare into vacancy in a somewhat excited manner, while all the time trying to do the things which were required of him. I myself had several interviews with him in which he seemed to be entirely rational, although it was with difficulty that I was able even temporarily to help him out of his condition of despondency. I made no attempt to hypnotize him, but I used every other psychic measure at my disposal to bring about a more cheerful and hopeful attitude with reference to his work. Just as I thought we were succeeding in our efforts, he suddenly, and without giving warning or notice either to me or to his hostess, packed his bag and went home. He immediately wrote me to the effect that he had decided to give up the treatment for a while at least, to which I replied that perhaps he would feel like returning later; but in about a week, I received a letter from his mother stating that her son had been greatly disturbed because he had not been able to more speedily overcome his speech difficulties, and although she tried in every way to cheer him up, he absolutely refused to be comforted, and after eating his breakfast one morning, he left his home, presumably to go to his place of business, but he went instead to a neighboring park and took his life with a revolver.

I mention this fact to show the awful mental suffering that sometimes attends this affection. I have had several patients tell me that self-destruction seemed to them at times to be the only real way out of their difficulties. This feeling is always increased with each failure to overcome the stammering, and in as much as about 90 per cent of those who attend the various institutions for the cure of the affection fail in reaching a solution of the problem of good speech, it is a question whether the schools for stammering on the whole may not be doing more harm than good.

Moreover, there is a common belief—and it seems to exist even in the medical profession—that children grow out of stammering, but this I think is a thing that never happens. It is true that the habit of stammering is often somewhat overcome in later life, but such improvement in speech is almost always the result of assistance from some source and great painstaking effort on the part of the individual. Children do not of themselves grow out of the affection, but they grow into it, and its awful consequences are well illustrated by the three cases I have described.

LI.

DIAGNOSIS AND TREATMENT OF DIPHTHERIA.*

By Joseph B. Greene, M. A., M. D.,

Asheville.

The fact that diphtheria has received such scant consideration from the laryngologists of the country is my reason for bringing this subject to the attention of this society. One cause of this apparent neglect is the fact that in many of our large cities patients with this disease are promptly transferred to contagious hospitals. This procedure develops a certain number of physicians in the hospital highly skilled in diagnosticating and treating diphtheria, and who are especially skilled in performing intubation and tracheotomy, which measures are at times so necessary for the saving of life. In consequence of this prompt transfer of patients, laryngologists of the cities see fewer cases, and are, as a result, called upon less frequently than formerly to perform intubation or tracheotomy. The early use of antitoxin has likewise materially reduced the number of cases requiring operation.

In our smaller communities the general practitioner still, to a large extent, attends his diphtheria cases alone, though I know of no disease where the internist and laryngologist can, to a better advantage to the patient, work hand in hand. The throat man can often be of service in determining the extent of the lesion, particularly as to the nose and larynx, and likewise diagnosticating early complications, as a beginning otitis media. The laryngologist, on account of his being accustomed to examine and treat the larynx, is better prepared to perform intubation when needed.

The diphtheria organism may attack any mucous membrane, and likewise the abraded skin, as in eczema and wounds. Diphtheria of the pharynx is the most common form, and the one to which I shall first direct your attention. Unfortunately, there is no method of diagnosticating, with positiveness, diphtheria from "ordinary sore throat" socalled, except by finding the characteristic organism, Klebs-Loeffler bacillus. Even when a culture is taken, one negative finding does not exclude diphtheria, if the clinical symptoms are suggestive, and in the absence of a positive finding, the patient should not be denied the benefit of antitoxin. The case which is reported later illustrates this point. The cause of a negative finding in cases of true diphtheria is not always the fault of the laboratory. It may be due to a failure to touch accurately the edge of the membrane, perhaps due to an imperfect view of the throat. One has often to contend against a struggling child, and frequently, too, in a poorly lighted room. It is also necessary to bring the inoculated part of the swab in contact with the surface of the blood serum. The use of antiseptics within an hour or so of the time of taking the culture destroys all chance of a growth on the serum tube.

---

*Presented as candidate's thesis at the nineteenth annual meeting of the American Laryngological, Rhinological and Otological Society, held at Washington, D. C., May 8-10, 1913.

The diagnosis of diphtheria has been relatively easy since Klebs, in 1883, discovered the organism in the diphtheritic membrane, and Loeffler in 1884, one year later, isolated the organism—hence Klebs-Loeffler bacillus. It is a small rod of irregular size and shape. It stains unevenly with Loeffler's alkaline methylene blue, giving at times a beaded appearance. This staining peculiarity is of great diagnostic importance, and is more marked in a culture twelve or more hours old than in a young culture, or from a swab of the throat. There is a tendency for the organism to assume a parallel arrangement, which has served me well in doubtful cases. This is more noticeable from a culture than from a swab. The organism is stained by Gram's method. As a differential stain, there is probably none better than Neisser's. Here the peculiar granules are stained dark blue, while the body of the organism assumes a light brown color. The most suitable culture medium is blood serum. On this it grows rapidly at body temperature, distancing the common pus organism, so, at the end of six or eight hours, a diagnosis can usually be made with the microscope. In some cases a staining of the swab will give valuable aid in the diagnosis, though a culture will be found more reliable.

Although the bacteriologic findings are of first importance in making a correct diagnosis, as before stated, when in doubt give antitoxin, if the case is at all severe. In primary laryngeal cases a negative finding is of little value if the swab is only taken from the pharynx. A laryngeal applicator could in these cases be used to advantage.

Cultures should be taken of all sore throats, particularly in infants and children. It is impossible to state otherwise, with accuracy, whether the case be one of simple tonsillitis or diphtheria. A correct diagnosis is important, not only for the safety of the child, but also from a public health standpoint. The following case will serve to illustrate two points—in the first place, the importance of not waiting for a positive finding before giving anitoxin, and secondly, the necessity of administering large doses of antitoxin.

Patient, L. C., age seven years. First seen by me at Asheville, N. C., in consultation with Dr. Powell, on Sunday night, July 2nd, the fifth day of her illness. The attack began with all the clinical appearances of simple tonsillitis. The culture negative. On Saturday, the day prior to my visit, the temperature was normal till 6:30 p. m., when it registered 99.5°. When I first saw her on Sunday night the picture had changed, for the membrane had spread from the right tonsil to the anterior pillar of the same side. A patch was also seen on the left tonsil. Temperature 103.5°. Neck very much swollen, and head extended to facilitate breathing. Patient showed signs of severe toxemia. Administered at once 10,000 units of antitoxin. The following morning patient no better; given 20,000 units of antitoxin. That afternoon, Monday, patient still dangerously ill. Culture positive for the first time. Seen with Dr. Dunn, and 40,000 units given. The next morning, Tuesday, seen with Dr. Briggs, patient slightly better, but still dangerously ill. Repeated 40,000 units. That afternoon patient much better, and only 20,000 units given. Small doses of antitoxin were continued for a few days, till all signs of membrane had disappeared. She was given, during her illness, 170,000 units of antitoxin. Her recovery was complete and uneventful, except for some paralysis of her palate and eye muscles. These had all disappeared at the end of nine weeks. Her pulse continued rapid for some time, which required absolute quiet. Stimulants were given as indicated.

Hoffman's bacillus closely resembles the diphtheria bacillus, though it is somewhat shorter and thicker. It does not show the polar bodies by Neisser, nor is it pathogenic to guinea pigs.

Bacillus xerosis,[1] as recently described, cannot be distinguished from Klebs-Loeffler bacillus. It is further said that bacillus xerosis forms acid from saccharose, not from dextrin; while bacillus diphtheria forms acid from dextrin, and not from saccharose. However, the final test in differentiating bacillus diphtheria from all socalled "pseudodiphtheria" bacilli is by animal inoculation. This slow method of differentiation can only be of use from a public health standpoint.

The clinical diagnosis of diphtheria is often difficult. It is particularly so in mild cases affecting the tonsils alone, and also where the larynx is primarily affected. Temperature is of little value in making a diagnosis of diphtheria. It is frequently not high, ranging from 99 to 100° F. Strange as it may seem, the temperature may be normal in cases of severe toxemia. A sudden rise in temperature suggests a beginning otitis media or pneumonia. The pulse, like the temperature, is a poor guide in the diagnosis, for it may vary from a slow pulse to a very rapid one. The extremes, as a rule, are of serious prognostic significance. The volume of the pulse soon becomes small, owing to a myocarditis. The depression is great in cases of severe toxemia, though in mild cases this need not be the case. It is too often said by the attending physician: "The patient is not sick enough for diphtheria." Follicular tonsillitis usually makes one seem sicker than true diphtheria. The temperature is usually higher. Before the appearance of the membrane there may be observed a peculiar plum color of the mucous membrane of the pharynx, particularly evident on the soft palate. There may be considerable edema present. Valuable time in the treatment may be saved by this early diagnosis. The membrane is at first of a grayish white color, though not unlike the membrane due to the pus organism. The cases of septic sore throat which occurred in Boston[2] from the milk epidemic of 1911 had some clinical resemblance to diphtheria. The bacteriology was different. The situation of the membrane is of great diagnostic importance. If found on the pillars, or any part of the pharynx other than the faucial tonsils, our suspicions should at once be aroused that we are not dealing with a case of simple sore throat. The odor in septic diphtheria may be foul. There is usually seen a peculiar red margin at the edge of the membrane, which has served me well for diagnosis in certain cases. The use of violence in removing the membrane to see if bleeding takes place is of little value, and is unjustfiable. Mucus in the throat may be gently swabbed away without harm to the patient. Albumin in the urine is suggestive of diphtheria. Early glandular involvement is a suspicious symptom of diphtheria, though the Boston milk epidemic of 1911 showed this symptom very frequently. There may be edema of the neck, which may extend down over the chest. This is strong evidence of diphtheria, and indicates a severe type of the disease. These are cases requiring large dosage of antitoxin.

Nasal diphtheria is the most contagious form, and from a public health standpoint is of the first importance. The membrane may be in the nose proper, or the nasopharynx. In the latter situation, that is, in the nasopharynx, the symptoms are more severe, owing to a rapid absorption through a bountiful lymph drainage. On account of the difficulty of inspecting the nasopharynx of very young children, the diagnosis of this type of diphtheria must usually be made from the symptoms and finding the characteristic organism. There is noted a serous or serosanguinous discharge from the nose, which may excoriate the upper lip. When the membrane is in the nose one can usually see it upon rhinoscopic examination. Frequently the membrane may be confined to one side of the nose, and then you get a one-sided discharge which simulates a foreign body in the nose. There may be nasal hemorrhage of some severity. The patient, if old enough, complains of a stopped-up feeling of the nose. The symptoms may persist for a long time, and may be so mild as not to be suspected as being diphtheria. As before stated, these cases are very contagious and become a menace to public health.

From autopsies performed by Councilman and Mallory at the South Department, Boston, it is probable that the sinuses of the nose, particularly the antrum and sphenoid, harbor the diphtheria organism for a long time. There may be an extension of the membrane from the nose to the ear, pharynx, larynx, or eye.

Diphtheria of the eye is a serious condition, particularly as to sight, and frequently results from an extension through the tear duct, though it may be due to accidental inoculation from other regions.

When the larynx is primarily involved it is difficult of diagnosis, especially in very young children. On account of the difficulty of inspecting the larynx in children by means of the indirect method, the laryngeal specula, as devised by Mosher and Jackson, are useful for direct laryngoscopy. The symptoms of laryngeal obstruction from a diphtheritic process come on slowly, and in this respect differ from a foreign body, and also spasmodic croup. At first there is hoarseness, followed by aphonia, evidenced in infants on attempting to cry. In a few hours this obstruction becomes greater, and we get signs of the patient breathing through a stenosed larynx, such as stridor, both inspiratory and expiratory in character, rigidity of the sternocleidomastoid muscle, and retraction of the abdominal muscles and supraclavicular spaces. There may be pallor instead of cyanosis, a fact which is easily overlooked when the question of giving relief by operative interference is considered. When the obstruction is lower down, as a foreign body in the bronchus or bronchial glands, there is more expiratory stridor. The auscultory signs of the chest in cases of foreign body are the absence of normal respiratory sounds over the area where the entrance of air is obstructed. When the obstruction is high up, as in retropharyngeal abscess, the stridor is more inspiratory in character. The diagnosis of retropharnygeal abscess can best be made by palpation with the index finger.

Catarrhal spasm of the larynx, or spasmodic croup, as it is sometimes called, may be mistaken for laryngeal diphtheria, but the former usually comes on suddenly at night, and there may be a history of previous attacks. The temperature is usually lower in spasmodic croup, though too much reliance must not be placed on this point in the diagnosis. Improvement usually follows in a few hours after appropriate treatment, not the case in laryngeal diphtheria unless relieved by operation.

Acute laryngitis in infants simulates diphtheria very closely. The constitutional symptoms are usually milder, except the temperature, which may be higher than in laryngeal diphtheria. Aphonia is not so marked. If there be a patch on the pharynx, glandular enlargement of the neck, or albumin in the urine, think of diphtheria. Direct laryngoscopy should be practiced in all doubtful cases.

Pneumonia resembles laryngeal diphtheria in the marked dyspnea, but there is usually no stridor. There can be elicited the physical signs of pneumonia, and probably a leucocytosis.

Jackson[3] has described a laryngotracheitis due to the influenza bacillus, which can be differentiated from diphtheria only by direct inspection. There is no membrane or Klebs-Loeffler bacilli present.

In distinguishing the laryngeal symptoms of measles, we should look for Koplik's spots, which appear before the skin rash. The eye and nose symptoms appear early in measles. A culture from the throat should, of course, be taken.

Diphtheria may be a complication of measles. In fact, it complicates this disease more frequently than any other.[4] Early laryngeal symptoms point to measles alone, while a later stenosis of the larynx in measles suggests diphtheria as a complication.

The diagnosis of diphtheria from scarlet fever, when a membrane is present, can only be made by the microscope. The general redness of the fauces, the appearance of the tongue, and perhaps the skin rash in scarlet fever may assist in the clinical diagnosis. One should be on the lookout for a complication of scarlet fever with diphtheria.

Vincent's angina has been very clearly and fully described by Place.[5] It may be mistaken for diphtheria. It is more chronic in nature, and usually shows a dirty patch on the tonsil or elsewhere in the mouth. Place says: "It differs from diphtheria in being always an ulceromembranous process, which diphtheria never is primarily." The finding of the bacillus fusiformis and the spirillum gracillis confirms the clinical diagnosis. Syphilis of the nose and pharynx may simulate diphtheria, but with the finding of the specific spirochete and the Wassermann reaction the diagnostic difficulties should be less.

Inherited syphilis of the nose in a young child may be mistaken for diphtheria, but there are usually other evidences of syphilis. It is less acute in character and there is the absence of Klebs-Loeffler bacilli.

Secondary syphilis of the throat is characterized by a thin layer of necrotic epithelia with a well defined border. There is usually less constitutional involvement, though there may be fever with syphilis.

Later forms of syphilis of the throat are characterized by a deep ulcer with a very much inflamed border. The appearance is out of all proportion to the symptoms, for there is usually little pain on swallowing. With the Wassermann reaction in syphilis and the finding of Klebs-Loeffler bacilli in diphtheria, there should not long exist doubt in the diagnosis of syphilis and diphtheria.

Traumatic exudates from operative procedures, as tonsillectomy, simulate diphtheria. A close inspection and a history of the case should remove all doubt. It should be remembered, however, that Klebs-Loeffler bacilli may be a complication of an operative procedure, so if there exist constitutional symptoms out of proportion to the appearance of the wound, a culture should be taken.

Leptothrix mycosis may be distinguished from diphtheria by its chronic course, often without symptoms. The projections on the tonsil are conical and show a growth of the leptothrix fungus.

In all lesions simulating diphtheria the microscope should be promptly used in clearing the diagnosis.

PREVENTIVE TREATMENT.

Patients should, if possible, be sent to an infectious hospital. In lieu of this, isolation and quarantine should be rigidly enforced at home. Immunizing doses of antitoxin, 500 to 2,000 units, depending somewhat on the age, should be administered to all exposed children. Adults should be carefully watched, as to their noses and throats.

There is hardly a question but that epidemics of diphtheria are kept up by "carriers," so, as a preventive measure, these cases should be sought for and isolated. Seligmann[6] lays stress on this point, and also speaks of nasal diphtheria being liable to convey the infection for a long period of time. Diseased tonsils and adenoids are especially liable to harbor the organism. The writer,[7] in 1908, removed diseased tonsils and adenoids for the purpose of getting rid of the diphtheria organism in a "carrier," and soon the patient was rid of the infection. Since that time this method has been frequently practiced in certain localities with marked success. It would not be advisable to practice this method as a routine procedure, but is suitable for certain selected cases. An immunizing dose of antitoxin should be given at the time of the tonsil operation, to prevent infection of the wound with Klebs-Loeffler bacilli.. Scheotz, a Danish physician, some years ago, recommended spraying the throat with a living culture of staphylococcus pyogenese aureus, to rid the throat of diphtheria infection. Page,[8] Lorenz and Ravenel[9] have practiced the overriding of diphtheria throats with staphylococcus pyogenes aureus, and report good results. As the two organisms are found growing side by side in throats, and can likewise be grown together in culture tubes, it is not easy to understand the reason for the disappearance of the diphtheria organism by this method of treatment. It would seem as if there is an element of danger in a spray of living staphylococci. Inasmuch as "carriers" have, as a rule, abnormal throats or noses, it would seem more rational to treat these abnormalities with a hope of ridding the patient of diphtheria bacilli. Place tells of a "carrier" who got rid of his infection when a foreign body was removed from the nose. At least two negative cultures, and probably three, should be required before release from isolation. The writer saw last year, while working at the South Department, Boston, a child who had been in the institution more than a year, awaiting the required negative cultures. Diphtheria vaccines[10] have been used with some success in ridding "carriers" of diphtheria bacilli.

SERUM THERAPY.

Under treatment, I shall consider first, antitoxin, for it is by far the most important. It is an absolute specific when given in time and in sufficient dosage. Of the 241 physicians, nurses, and attendants contracting diphtheria at the South Department, Boston, not one succumbed. It is not necessary to consume your time in offering proof of the efficacy of antitoxin in this formerly much dreaded disease. I assume that every member of this society is a believer in the life-saving value of antitoxin. It should be given early, and in sufficient dosage to produce the desired effect. The size of the initial dose should vary from 5,000 to 50,000 antitoxin units, and more in the malignant cases which have existed for some time. As McCollom rightly says, the age, over two years, has nothing to do with the size of the dosage. It is a chemical reaction, and not a physiologic one, so there is no reason to doubt but that a young child may have as many toxin units as a fully grown man. They certainly have a lessened resistance on account of their tender years. The antitoxin is, in no sense, an antiseptic, but in neutralizing the toxin it gives the leucocytes and other protective forces a chance to get in their work.

Textbooks have been very inadequate in the size of the dosage recommended. Tyson, in his "Practice of Medicine" recommends 1,000 to 2,000 units, depending upon the severity of the attack. Ballenger recommends 2,000 to 3,000 units in what he terms ordinary diphtheria; 3,000 to 5,000 units in laryngeal cases. McCollom, in his classical paper on diphtheria in Osler's Modern Medicine, says: "If there is a very extensive membrane when the patient is seen for the first time, 8,000 to 10,000 units should be given, repeated every four or six hours, until the characteristic effect of the serum is produced, namely, the shriveling of the membrane, diminution of the nasal discharge, a correction of the fetid odor, and general improvement."

Since the above was written, the dosage at the South Department, Boston, has been increased, and the mortality likewise reduced. It may be of interest to state that the mortality at this large infectious hospital, from 1888 to 1894, the year of the introduction of antitoxin, was 43.2 per cent. From 1895 to 1904 inclusive, it was 11.48 per cent. For the year 1912 it was 7.6 per cent, including laryngeal and moribund cases. The greatest reduction was in the very severe cases where massive doses of antitoxin were administered.

Both nasal and laryngeal diphtheria require large doses of antitoxin. Abraham Levinson[11] says that the curative dose in nasal and laryngeal cases should not be less than 25,000 units. Diphtheria of the eye, on account of the danger to sight, requires large doses of antitoxin. It is far safer to give larger doses than is required to neutralize the toxin, which is so destructive to the vital cells, than to leave a portion free to exert its deadly effect on the tissues of the body. There is no way of estimating, with accuracy, the amount of toxin requiring neutralization, but the amount depends somewhat upon the duration of the illness, the extent of the surface involved, and, lastly, the virulence of the organism. A mild case of pharyngeal diphtheria should receive, as a single dose, from 5,000 to 10,000 antitoxin units; a moderately severe case of several days' standing should receive 20,000 to 25,000 units, and a severe case, 50,000, and more if the case seems desperate. The size of the dose can soon be reduced and smaller doses continued until the membrane has completely disappeared. Place strongly advises getting all the required antitoxin in during the first three days of the treatment, even if 500,000 units are required. This dosage, of course, refers only to the desperately ill cases.

Ehrlich says two poisons are produced by the growth and multiplication of the diphtheria bacillus. To one he gives the name of toxin, and to the other toxon. To the former he attributes the early acute symptoms, and to the latter, the toxon element, he attributes the late paralyses. The affinity is stronger between the toxin and antitoxin than between the toxon and the antitoxin, hence the importance of having a surplus of antitoxin to neutralize the toxon element after the toxin has been chemically disposed of.

There is what is termed a precocious form of palatal palsy occurring about the fifth day of the disease. This is not so serious as the late forms, occurring usually within the first three weeks of the illness, but the early form is somewhat an index to the severity of the illness.

Rosenau and Anderson[12] have shown by animal experimentation the uselessness of giving antitoxin with a hope of influencing the paralyses occurring late in the disease.

My favorite situation for injection of the antitoxin is in the loose areolar tissue of the back, near the angle of the scapula. This region is relatively free from the sensory nerves, is protected from vital parts, and lastly, the operation is not witnessed by the patient. This last point is quite important from a psychic standpoint. The site selected should be sterilized, and the situation should be changed from time to time, as there is usually a local reaction.

Fritz Meyer[13] strongly recommends intravenous injection of antitoxin in serious cases—says much time is saved in the absorption. Eckert[14] and Hoesch[15] and others advise intramuscular injection as being more rapid of absorption than the subcutaneous method.

DANGER OF ANTITOXIN.

The danger of antitoxin has been grossly exaggerated. That a few deaths have taken place following its administration cannot be denied, but they are too infrequent to be seriously considered in view of the dangers of diphtheria when left untreated by antitoxin.

Park[16] says that "In over 100,000 patients immunized (New York) since the introduction of antitoxin, there has been but one known fatality due to the serum injected. This child suffered from status lymphaticus and died shortly after an injection of 1000 units." Place says that more than 100,000 doses of antitoxin have been administered to over 40,000 patients at the South Department, without a dangerous symptom attributable to antitoxin.

The work of Theobald Smith, followed by the work of Otto, Rosenau and Anderson,[17] proved that horse serum administered to guinea pigs sensitized them, after an elapse of about ten days, to the same kind of serum. A small second dose was often sufficient to cause death with symptoms of distressed respiration, cyanosis, convulsion and finally death. This phenomenon has been termed anaphylaxis. While this reaction is common in laboratory experiments with guinea pigs, it is very rare clinically. It would be a great pity if these interesting laboratory experiments should curtail the use of antitoxin in diphtheria. It has been thought that the rare appearance of severe symptoms following the use of the first antitoxin dose is due to a natural "sensitization," or anaphylactic condition. The symptoms are marked respiratory distress, and may be due to the contraction of the smaller bronchi. Asthmatics, hay fever patients, and those sensitive to the emanations from the horse are more liable to have symptoms of anaphylaxis on administration of antitoxin. Adults seem to be more subject to this condition than young children. It would seem, if such patients require antitoxin, that they should be given a preliminary dose of atropin, followed by a tentative dose of antitoxin. The writer resorted to this procedure on one occasion where the patient had experienced severe respiratory disturbance following a dose of antitoxin in a previous attack of diphtheria. The patient was given a full dose of atropin, hypodermatically, just fifteen minutes prior to the antitoxin.

Concentrated antitoxin was then administered in small doses every fifteen minutes until the required amount was given. In patients in whom there is a suggestion of a sensitized state, this method is worthy of a trial. It may be of interest to analyze the sixteen deaths reported in the literature following the use of antitoxin. The size of the dose in each case was small, varying from 500 to 4,000 units. Only six of the sixteen cases were suffering from diphtheria, and six others, owing to exposure to diphtheria, were given small immunizing doses. The remaining four did not have diphtheria at all, but were given antitoxin for asthma. Nine of the sixteen gave a history of asthma in some form.

There is another condition known as "serum sickness" following at a variable interval the administration of antitoxin, usually about the tenth day, characterized by rashes of various kinds, with intense itching, joint pains and fever. There may be some edema. The pulse is quickened, but of good strength. This condition may at times be mistaken for measles or scarlatina, though there are not the usual symptoms of these diseases. Park says that about 20 per cent develop the rash. The reaction is certainly less in the concentrated serum where the irritating proteids have been removed by the Gibson process than in the more bulky product. Calcium chlorid[18] has been strongly recommended in gram doses, beginning on the day of the injection of antitoxin.

The treatment of serum sickness is largely symptomatic. Sponge with cold water for the fever, menthol solution for the itching, and heat to the swollen joints.

The general treatment of a patient with diphtheria should be the same as other acute infectious diseases. The room should have abundance of fresh air. The food should be light and nourishing. The urine should be examined occasionally. On account of the heart bearing the brunt of the attack, it should be guarded as much as possible.

The patient must be kept quiet in bed, not only during the attack, but for a considerable period of time after the illness, depending somewhat upon the severity of the attack. The time should also be longer in cases where antitoxin was given late.

Cardiac stimulants should be given as the heart shows signs of weakness. This can best be determined by listening to the heart sounds, particularly the second pulmonic. Jacobi[19] has always been a great advocate of alcohol in some form. At the South Department, Boston, reliance is placed on camphor, caffein, atropin and strychnin.

Cracked ice held in the mouth is grateful, and ice packs to the neck are useful, particularly where edema is present.

The use of local antiseptics is of secondary importance, and if strong may be actually harmful. In my judgment, anything stronger in the nose than normal salt solution or liquid vaselin is not to be recommended. To the pharynx in young children antiseptics are of doubtful value, as the physical resistance offered may be more harmful than the local treatment is useful. In older children and adults, my favorite application is a solution of one part of peroxid of hydrogen to three or four of water. Some advise Loeffler's solution, while others prefer irrigation with a hot normal saline solution. The ear should be watched for a beginning otitis media.

Paracentesis should be performed early. Borden[20] lays stress on the number of mastoid involvements discovered at autopsy in patients dying with diphtheria whose drum membranes were apparently negative.

Pneumonia is a serious complication, and is especially frequent in laryngeal cases. Stimulants should be given as the case requires.

Gallop rhythm is a serious complication or sequela, and is best treated by absolute rest, rectal feeding, and appropriate stimulation.

Diphtheritic paralysis is a frequent sequela of diphtheria. It may attack any nerve, but particularly those of the palate and larynx. It is more serious when the pneumogastric is involved.

In laryngeal diphtheria, particularly in intubation and tracheotomy cases, it is advisable to use steam inhalations under an improvised tent. Ipecac may be used in slight stenosis pending the action of antitoxin. This should be relied upon but for a brief period of time, for we must remember that the laryngeal stenosis not only taxes the respiratory system, but gives an added burden to a heart already weakened by the diphtheritic toxin. As before mentioned, these are cases requiring large doses of antitoxin.

In intubation cases the question of feeding comes up. As a rule semisolid food is best, and should, if possible, be given in the upright position. Rarely is it necessary to use a stomach tube or the Casselberry position.

Whether intubation or tracheotomy is preferable in laryngeal stenosis of diphtheria has long been a mooted question. Since the introduction of the intubation tubes by O'Dwyer, of New York, which marks one of the brightest spots in American medicine, physicians on this side of the waters have shown a decided preference for intubation. France has for a long time been an advocate of intubation, while Germany is using more and more the O'Dwyer tubes. England, in her conservatism, clings to tracheotomy. It is not to be understood that intubation is to entirely supplant tracheotomy, for there are certain conditions where tracheotomy is preferable.

The advantages of intubation are:

1. Relief of the stenosis without the shock of a cutting operation, and without an anesthetic.

2. Consent of the family for operative relief is more readily obtained. This is a great advantage, as early relief of the stenosis is a great factor in reducing the mortality.

3. There is no danger of local infection, and less danger of pneumonia in intubation than in tracheotomy.

4. There is no resulting scar.

Tracheotomy instruments should always be at hand where intubation is performed, in case of stoppage of respiration from pushing the membrane in front of the tube. This is not a frequent occurrence. Rarely the patient persists in coughing up the tube, which, if continued, requires tracheotomy. There are also cases where there is marked pharyngeal swelling, together with edema of the aryepiglottic folds, where tracheotomy is indicated. Lastly, if the surgeon cannot remain within easy reach of the patient, in case of stoppage of the intubation tube, or its coughing up, it might be safer to perform tracheotomy. It does not seem necessary to give the technic of intubation or tracheotomy, for excellent descriptions of these operations are given in our textbooks. However, the ingenious method of Mosher's[21] intubation deserves mention, and in the hands of some should prove simpler than the indirect method. The same O'Dwyer tubes are used, only it is performed with a straight introducer, through a laryngeal speculum. If the direct method is used for diagnosis, it might be best to continue the operation and insert the tube with Mosher's introducer.

The writer wishes to emphasize the following points:

1. The diagnosis of diphtheria depends primarily on finding the organism. Cultures should be taken of every sore throat in children, and likewise suspicious nasal discharges. But in the absence of a positive finding, if the symptoms point to diphtheria, antitoxin should be given. This is particularly important if the symptoms are severe.

2. Much larger doses of antitoxin should be given in diphtheria. Textbooks have been misleading in this respect.

3. Laryngeal cases are serious partly from mechanical obstruction, requiring prompt relief of the stenosis, and large doses of antitoxin. Intubation in the main is preferable to tracheotomy. Laryngologists should perfect their technic in this operation until it becomes a fairly simple one.

4. Epidemics of diphtheria are kept up largely by "carriers." They should be sought out, isolated, and the abnormality treated. Diseased tonsils and adenoids may require removal.

REFERENCES.

1. Hiss and Zinser's Bacteriology.
2. Darling: Boston Medical and Surgical Journal, December 14, 1911.
3. Jackson: Laryngoscope. Editorial, February, 1912.
4. McCollom: Osler's Modern Medicine, Vol. II.
5. Place: Boston Medical and Surgical Journal, December 14, 1911.
6. Seligmann: Zeits. f. Hygiene, Vol. XX, 1911, p. 68.
7. Greene: Laryngoscope, August, 1908.
8. Page: Archives of Internal Medicine, January, 1911.
9. Lorenz and Ravenel: Journal A. M. A., January 22, 1912.
10. Editorial, Journal A. M. A., September 14, 1912.
11. Levinson: New York Medical Journal, January 6, 1912.
12. Rosenau and Anderson: Hygienic Bulletin No. 38.
13. Fritz Meyer: Berlin klin. Wochen., June 28, 1909.
14. Eckert: Handbook of Serum Therapy.
15. Hoesch: Deutsch. Med. Woch., 1911, p. 1683.
16. Park: Journal A. M. A., February 17, 1912.
17. Rosenau and Anderson: Hygienic Bulletin No. 29.
18. Nuttall and Graham: Smith, Diphtheria.
19. Jacobi: Treatise on Diphtheria.
20. Borden: Transactions Ninth International Otological Congress.
21. Mosher: Laryngoscope, September, 1910.

LII.

TWO CASES OF PERFORATION OF THE PETROUS PORTION OF THE TEMPORAL BONE DUE TO THE CAPSULATED BACTERIA.

By H. B. Graham, M. D.,

San Francisco.

In the Annals of Otology, Rhinology and Laryngology for December, 1910, I made a short review of the work that had been done on the capsulated bacteria in the acute middle ear infections. Since then I have had the misfortune to operate two cases which illustrate the insidiousness and burrowing power of this group of organisms. I append the two histories and photographs of the temporal bones.

Case 1.—M. D., age 50 years, came to the clinic February 19th, with a history of having had a severe bronchitis in January. About February 5th had had a dull, left-sided, intermittent pain in the head with hissing noises and deafness in the left ear. No discharge. Examination at the clinic gave a right temporal headache, pain over left ear, with a marked external otitis. The patient did not return to the clinic until March 3d, when I saw him for the first time. At this time there was a subperiosteal abscess which probably came from the mastoid. He complained of pain over left eye and of slight dizziness. There was no nystagmus. He was operated March 4th. I uncovered the sinus and dura in posterior and middle fossæ and the whole mastoid was removed. The temperature, both before and after the operation, for two days ranged between 98° and 99° F. On March 7th the patient was feeling well, the pain and dizziness had left, and he was permitted to leave the hospital. On March 8th he was taken to the City and County Hospital unconscious, with a temperature of 102° and pulse 120. On March 10th exitus.

Autopsy Findings.—(Dr. Ophüls.)—Walls of the mastoid cavity were made up of apparently normal bone. At the base of the cavity, near middle ear, were a few drops of thick yellow pus; postmortem clots and liquid blood in the longitudinal sinus. Meningitis purulenta at base of brain. Underneath the tentorium, about 1 cm. laterally from internal auditory canal, there is a hyperemic spot in the dura about 1 cm. in diameter with a central grayish discoloration; underneath the dura there is a large ragged opening in the bone about 1 cm. in diameter; there is a small accumulation of pus between the bone and dura; at the bottom of the cavity there is an opening in the bony capsule of the posterior semicircular canal. In the middle ear there is mucus and pus; the mucous membrane is swollen and hyperemic. From the middle ear a fistulous opening passes through the temporal bone and appears on the posterior surface at the spot where the abscess underneath the dura was found. Smears from the middle ear and meninges show a very small diplococcus resembling the pneumococcus.

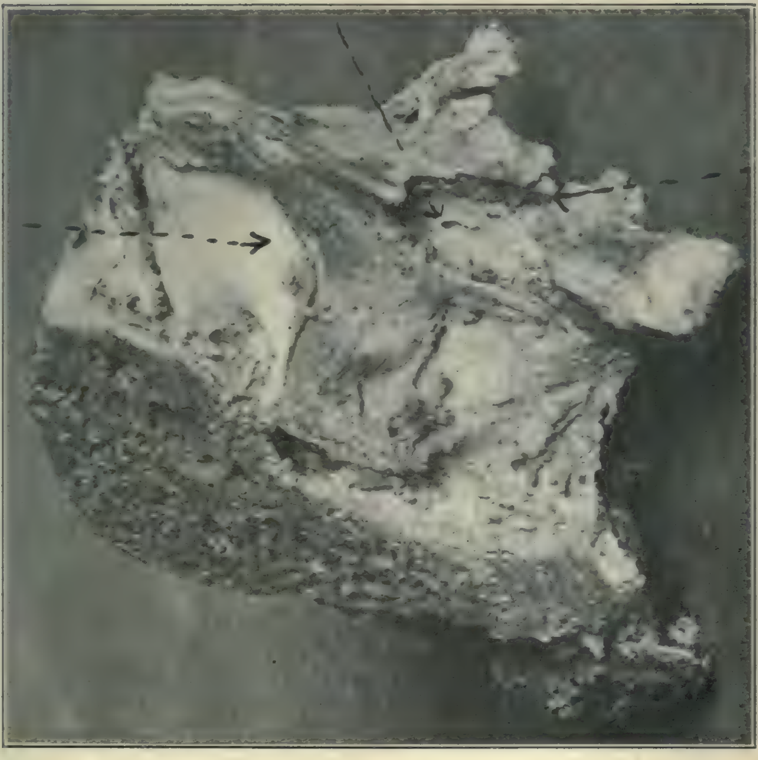
In this case the initial bronchitis, followed by a period of indefinite pains in the head with symptoms referable to the ear and a low temperature, apparently over a long period of time, would have led to an earlier diagnosis and operation if the patient had not been so irregular in attendance and had been more communicative when in the clinic. That this man carried a meningitis about with him for days before he died or was even operated can hardly be doubted from the appearance of the meninges at autopsy, and still he had but few symptoms that were referable to the meninges at the time of operation. The meningitis due to these capsulated bacteria is an extremely quiet one and can very easily be overlooked by the most careful observer.

Case 2.—A. C., age 49 years, steward, Scotchman. Entered clinic October 10, 1912, complaining of pain over the right ear and extending to the top of the head. There was a large amount of pus in the nose and an ethmoid operation was done. Following this the patient disappeared, and entered the clinic again October 30th, stating that he had had a pneumonia and pleurisy. I saw the patient for the first time on October 30th, when he had an abscess behind the ear. I operated, finding pus under the periosteum and a fistula under the anterior spinous process. The longitudinal sinus was covered with granulations. The temperature was normal after the operation and the patient supposedly made an uneventful recovery, going to work with a small opening behind the ear. On December 6th the patient was readmitted to the hospital with well-marked signs of a meningitis. Lumbar puncture showed a turbid fluid with shreds. The temperature rose rapidly from 101° to 104°, pulse from 90 to 150, and exitus in December.

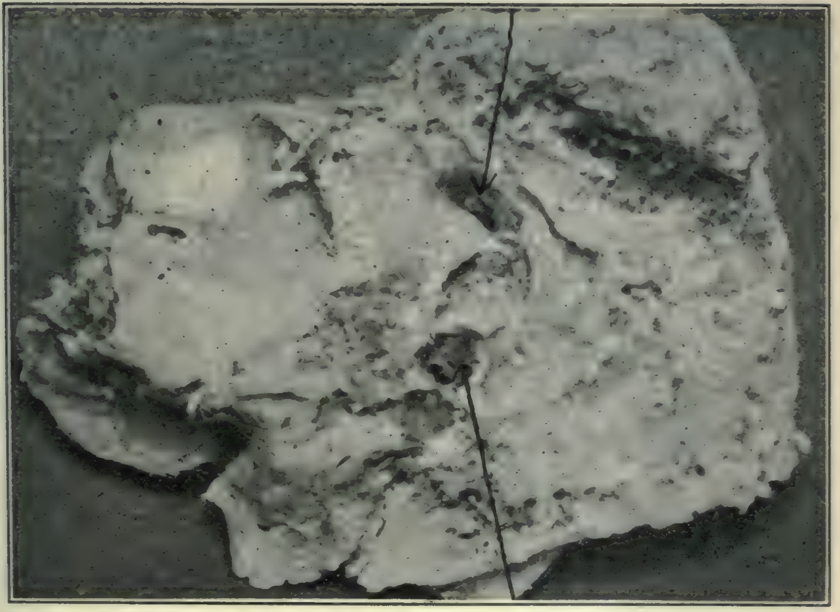
Autopsy Findings.—(Dr. Ophüls.)—Scar from old mastoid operation on left side; opening from a recent operation on the right. The meninges are covered with a thick purulent exudate. On lifting the brain from the petrous portion of the right temporal bone there is seen an opening in the bone at the lateral edge of the temporal ridge about 1 cm. in diameter and one-half cm. deep, which is somewhat crescent shaped. A little cavity is formed by the defect between the dura and the bone, in which there is a small amount of pus. This cavity connects by a fistula with the middle ear at a point on the floor in front of the eustachian orifice. The bone and under surface of the dura are covered with granulation tissue. The middle ear contains a mucopurulent fluid, and pale granulation tissue covers the small bones. The operated area consists of grayish red granulation tissue several millimeters in thickness. There is no pus in the internal auditory canal. Left middle ear normal. Left frontal and right and left ethmoidal sinuses contain a small amount of mucopus. There is a syphilitic atheroma of the aorta. Smears from the wound, meninges, lateral ventricle, middle ear, subdural abscess and lung show many polynuclear leucocytes and many encapsulated diplococci and short streptococci and streptococcus mucosus.

In this case the fistula had taken a course diametrically opposite to that in the former case, the one appearing in the posterior, the other in the middle fossa.

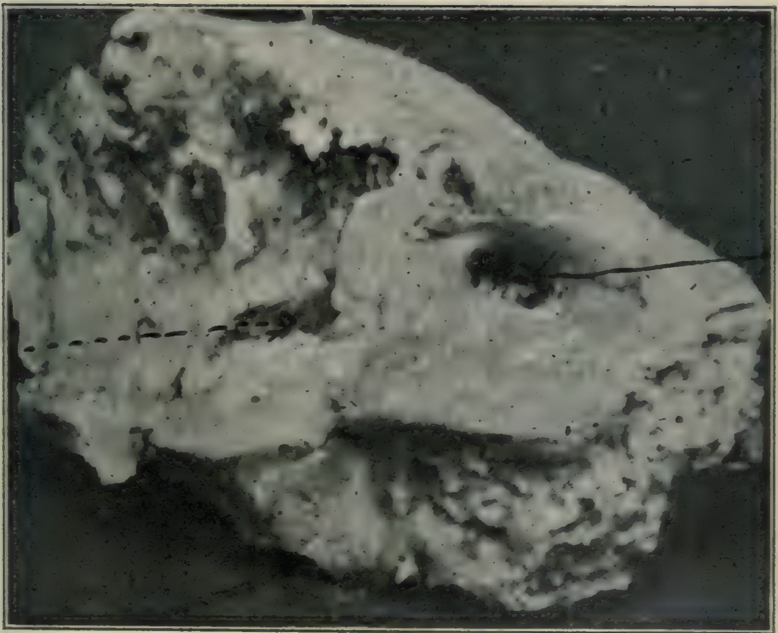
The accompanying illustrations are self-explanatory.


Posterior semicircular canal.
ternal
ditory
nal.
Fistula
from
antrum.

CASE 1.



CASE 2.



CASE 2.

LIII.

THE SURGICAL MANAGEMENT OF DISEASES OF THE FRONTAL SINUS.

By Robert Cunningham Myles, M. D.,

New York.

The problem as to the best method of surgical procedure in each case of frontal sinus disease has created many anxious moments of serious deliberation for the conscientious surgeon. The cause, process, and pathologic stage are important factors to be considered in our plan of management.

Cases of acute frontal sinusitis with obstructive symptoms should be controlled by strict discipline for the patient, and by prompt, watchful, active, and definite procedures on the part of the attending surgeon. The patient should be ordered to rest, with a minimum of physical exercise. Temporary and repeated applications of solutions of adrenalin chlorid and cocain, used on a small cotton applicator, under the anterior end of the middle turbinal and within the nasofrontal canal, will frequently relieve the ominous and threatening destructive pressure within the sinus. If this fails, the operation for the removal of the anterior end of the middle turbinal should be performed, and drainage of the sinuses secured by the dilatation probe or by removal of a part of the anterior ethmoidal cells and the projecting portion of the nasal process of the superior maxillary bone. The importance of limiting the operative area as much as is consistent with securing sufficient drainage should always be carefully considered, on account of the possibility of spreading the acutely infective process. The writer believes that we would be justified in making a temporary drainage externally above the inner canthus of the eye, in case of failure of attempts to relieve the disease intranasally, because he believes that it is wiser and safer to delay the performance of the radical operation for about three weeks in order that the usual virulence of the infective bacilli may become exhausted.

The proper management of the acute and subacute cases of frontal sinusitis will lessen the number of chronic cases by a large percentage. There are many classes of types of cases of chronic frontal sinusitis demanding a nicety of individual judgment, and when we consider the responsibility assumed by the authority of our judgment, we are well aware of the hazard encountered in making statements as to rules for definite methods of procedure. Our science has not advanced far enough to warrant unerring rules of accuracy in all cases, but it is with pride that we notice the wonderful improvement which has been made in the treatment of the diseases and in the operative technic since the early days when the writer was so actively struggling in the mystic meshes of these complex cases.

It is evident that the pendulum swings too far with all of us in regard to the internal or the external operation, and it is also evident that habit or bias influences our methods of procedure when a cold analysis of symptoms and physical conditions should determine the best plan of surgical management. Very serious conditions often require desperate remedies. We should be prepared to do a complete radical operation when there is considerable necrosis of the mucous membrane, periosteum, or of the bone, with or without perforation of any of the walls. When there is a neoplastic growth, especially of a malignant nature, an external radical operation is necessary, though there may be an exception in some cases of syphilis. The very small sinuses can be managed by the open method through the floor beneath the orbital ridge, without much facial deformity.

The Killian operation is the best method of procedure in cases of serious disease of the large sinuses, especially where previous injudicious and incomplete operations have been performed. Sometimes a partial Killian, using his bridge with the open method, will afford the best results in certain types of cases of the latter class. Since the writer many years ago adopted the habit of removing the anterior end of the middle turbinal, most of the ethmoid cells, and a part of the nasal process of the superior maxillary bone, he has "managed himself out of" an opportunity to operate externally in over 90 per cent of his cases.

The use of drainage tubes from the sinus to the nose has been abandoned after the performance of internal operation, because they seemed to excite that granulation tissue which is nearly always the cause of the failure in our unsuccessful cases. The writer has always condemned semi- or thorough curettage of the sinus where we do not do the complete obliteration operation. Free drainage and free ventilation are far better for an edematous and infiltrated mucosa than a surgical mutilation. Irrigations with normal salt solution or a solution of boric acid, passed up through the enlarged nasofrontal duct with the author's malleable silver canula, have proved to be of valuable service in bringing about a favorable result. Filling the cavity with solutions of argyrol, iodoform or vaselin, or Beck's paste has, at times, apparently overcome very stubborn pathologic conditions.

In conclusion, the writer would advocate certain rules for the surgical management of the diseases of the frontal sinus.

1. When the acute cases have unbearable, severe, and continuous pain in the sinus and above the eyes, attended with profound constitutional symptoms—fever, chills, and depression—we should proceed in a definite manner to relieve the pressure, if necessary, by doing a submucous operation on an obstructive deflected septum and by removal of the anterior end of the middle turbinal, a part of the anterior ethmoidal cells, and the nasal process of the superior maxillary bone; and, if this fails, should make a small incision in the skin above the inner canthus of the eye and a small opening in the bone—a radical operation to follow a few weeks later, if necessary.

2. Chronic cases should be treated by the external operation where there is evidence of bone necrosis, either with or without perforation; for neoplasm, especially of malignant nature; for meningeal symptoms which are probably due to sinus disease; for continued attacks of severe pain and discharge mixed with pus and decaying débris; and to satisfy patients who demand relief from recurring attacks of frontal sinusitis after failure to obtain relief by the internal operation.

The Killian operation, combined with removal of the anterior ethmoidal cells and nasal process of the superior maxillary bone, has proved to be a most satisfactory method in cases of large sinuses. The drainway into the nose from the space where the sinus had been should be kept open by tubes until the open space under the bridge has become obliterated. Compression over the sinus will aid materially in hastening the healing. The use of the artificial blood clot—either combined with paraffin or not—will decidedly relieve many of the objectionable deformities.

LIV.

REPORT OF A CASE OF ULCERATION OF THE LARYNX; PERICHONDRITIS OF THE ARYTENOID CARTILAGES, ABSCESS AND PARTIAL EXFOLIATION OF BOTH CARTILAGES RESULTING FROM TYPHOID FEVER.*

By J. H. Bryan, M. D.,

Washington, D. C.

This case is reported in order to emphasize the importance of making regular and systematic examinations of the upper air passages, especially of the larynx, in all cases of typhoid fever, in order to detect the early changes that take place in the mucous membranes of the upper air passages in this disease.

On the night of October 12, 1912, I was called to see Major C., U. S. A., aged forty years, at the Walter Reed Hospital, suffering from a severe case of typhoid fever, then in about the fiftieth day of the disease. I was called to see the patient because of his difficulty in breathing and loss of voice. The following history was given me by the attending surgeon: In September this officer returned to his duties in Washington, after a western trip, suffering from typhoid fever. The disease ran rather a severe course from the beginning, with high temperature and several severe intestinal hemorrhages. The temperature gradually subsided, and within a week or ten days of my seeing the case he was supposed to be convalescing. About four days before I was called to see him, the patient was thought to have taken cold, a slight laryngitis developed, accompanied by a cough and a husky voice, but at no time did he complain of any pain in the region of the larynx.

When I saw the patient, he presented a very emaciated appearance, with an anxious expression of the face, skin bathed with sweat, respiration rapid and shallow, temperature 100°, pulse small, thready, and rapid, and voice husky. Laryngoscopic examination showed fauces very much congested, but no ulceration in this region. There was extensive swelling of the right ventricular band, which overlapped and partially obscured the corresponding true vocal cord, which was in the median line and did not move on deep inspiration; there was also seen a deep ulceration on the under surface of the ventricular band, which seemed to extend to the free margin of the true vocal cord. Both arytenoid cartilages were markedly swollen and posteriorly were bathed in a thin, grayish secretion, probably purulent in character. The left vocal cord moved somewhat on inspiration, leaving a narrowed glottis, sufficient, however, to admit of respiration.

---

*Read before the American Laryngological Association, Washington, May, 1913.

The gravity of the situation was explained to the family and a tracheotomy advised, but owing to the fact that he seemed to be breathing deeper and was more quiet, I left him about midnight, cautioning the surgeon in charge to do an immediate tracheotomy if his respiratory difficulty increased. I saw the case again the next morning at ten o'clock, and while the patient had passed a restless night, he seemed, on the whole, somewhat improved as to his local condition, but not so well generally. The examination of his larynx showed the condition about the same as when last seen at midnight, with the exception that the excursion of the left vocal cord was greater, giving a much wider chink to the glottis and thereby admitting of freer respiration. The consent of the family having been obtained, it was arranged that I should return to the hospital at two o'clock, when a tracheotomy would be done. I had not been away from the hospital more than an hour when the patient had a severe coughing spell which brought about a spasm of the larynx, necessitating an immediate tracheotomy, which was done by Captain Smith, the medical officer in charge of the case. The patient did not breathe after the trachea was opened, and died almost immediately, although stimulation and artificial respiration were resorted to. Owing to the prolonged illness, the patient's heart was greatly weakened, and his death was due to heart failure about the time the trachea was opened.

The larynx was removed, and unfortunately placed in Müller's fluid, which discolored the specimen. The following day

Major Russell of the Army Medical School and I opened the larynx and found the following conditions: The right ventricular band swollen, and a large ulceration on its under surface; the true vocal cord ulcerated on its free margin; both arytenoid cartilages completely detached and lying loose in their capsules, the right cartilage being held in position by only a small membranous attachment. A bacteriologic examination made by Major Russell proved negative.

PHOTOGRAPH OF SPECIMEN.

The photograph of the specimen does not show the destructive changes that were found in the larynx as clearly as the natural specimen—the shadow is so deep that the right arytenoid cartilage is seen with some difficulty—but it is sufficiently distinct to enable us to see the destructive changes that have taken place.

In the light of what has been accomplished by antityphoid vaccination in the United States army, it may be asked why this officer had not been immunized. Several years ago he contracted pulmonary tuberculosis, and for a while was under treatment for this disease at Fort Bayard, but later was discharged cured and returned to duty in Washington. In view of his previous tubercular history, and as tuberculosis is a well recognized contraindication to typhoid vaccination, it was deemed unwise to subject him to this treatment.

This brings up a very interesting point in connection with typhoid prophylaxis, and that is, that typhoid vaccine acts very much in the same way as tuberculin, with a constitutional reaction and fever lasting several days. Some French surgeons claim to have had cases in which latent tubercular lesions have been excited and made quite evident after typhoid vaccination.

Owing to the insidious character of the development of this complication, it is wise that a laryngeal examination should be frequently made of typhoid cases, especially so when there is the slightest change noted in the character of the voice, cough, or difficulty in respiration. Jackson,[1] in a very interesting article on this subject, after a study of 360 cases, quotes Dr. John W. Boyce on this point, who says it is a question of great interest whether it is possible for these affections to cause a fatal termination without such symptoms as shall plainly indicate the nature of the occurrence. He further says, theoretically it seems impossible that a man should choke to death without the conspicuous symptoms of asphyxia, particularly cyanosis, but observation has convinced him that it is only too true; that these cases may sink away as peacefully as a baby goes to sleep, and that one may not in the least suspect what is happening.

Jackson divided his cases into subacute catarrhal laryngitis, ulcerative laryngitis, and perichondritis. The first was found to be present in 227 cases (or 63 per cent), the second in 68, and the third in 17, of the 360 cases.

St. Clair Thompson,[2] in the chapter on laryngeal complications in typhoid fever, in his work on "Diseases of the Nose and Throat," gives the frequency of occurrence from the European standpoint as follows: Landgraf, 11 per cent of all fatal cases; Griesinger, 26 per cent of all fatal cases; Kanthack, 26 per cent of all fatal cases; Ouskow, 30 per cent of all fatal cases.

It is difficult to arrive at any conclusion as regards the comparative frequency of this complication of typhoid fever in this country and abroad. The figures given by Jackson seem to show that a much larger number of cases of laryngeal involvement occur in this country than is indicated by the figures given by Thompson. The epidemic in which Jackson made his observations, however, was an unusually severe one, and his subjects were largely of a poorly nourished type, and this may account for the apparently greater frequency of this complication in this country.

We will not be able to get at the truth of this matter until more careful observations are made, not only in the hospitals, but in private practice as well.

REFERENCES.

1. Jackson: Journal American Medical Association, November, 1905, page 845.
2. St. Clair Thompson: Diseases of Nose and Throat, page 671.

LV.

VASOMOTOR DISTURBANCES OF THE UPPER AIR TRACT.

By C. W. Richardson, M. D.,

Washington.

During the past few years it has been my observation that the rhinologist is beginning to appreciate the fact that the upper air tract, especially the nasal tract, is not an independent organ of the body, but that it is part of an organism and is influenced pathologically, to a large extent, by disturbances in other organs and systems of the body. Acting upon this gradually increasing impression among rhinologists, members of our specialty are looking, or beginning to look, beyond the nose to determine the cause of and remove the conditions which bring about various diseased activities within the nasal chambers. One is impressed with the increased frequency with which very excellent and timely papers are appearing in the journals bearing upon and elucidating points treating upon this corelationship of the nasal organ and the bodily condition, evidenced by thought, research and keen study, often with reports of cases bearing upon some important type of interrelationship. My only excuse for reciting the few words I shall bring to you is that the subject is such an important one that it should be furthered in every way that our feeble insistence can carry it. One is impressed with the decreasing frequency with which intranasal operative work is now being done for vasomotor disturbances of the upper air tract. Some years ago it was the common procedure to cauterize and remove turbinates for this disturbance. I regret to note that, even at the present day, this type of surgery is too much indulged in in efforts to relieve this condition. It has been my experience that this form of nasal disorder, if thoroughly studied from its general aspect and those conditions which are instrumental in provoking it, can be relieved or removed and a moderate degree of simple nasal treatment instituted, in which surgery plays no part, a resolution with permanent return to the normal can be assured.

Only a few months ago I discharged from my care a case exemplifying this condition, upon which a submucous operation for restoration of a deflected septum had been done. The operation, as far as I could see, had been attended with a perfect operative result, but the patient had never received any beenfit so far as his breathing was concerned. The septum was perfectly straight, but the sodden, swollen turbinates filled in all the breathing space. Proper instruction as to diet, exercise and avoidance of overwork and excessive nervous zeal, with mild local treatment, resulted in a rapid improvement and later a complete restoration of the normal lumen of the nasal cavities.

Another type of case which I fear also the rhinologist has too lightly considered in the past is the large group of cases wherein there is more active congestion of the turbinates, with intermittent turgescence of varying degrees. Close inquiry in many of these cases will demonstrate that the disturbance is brought about and maintained by faulty metabolism and autointoxication. All methods of local treatment of a surgical character employed to relieve such conditions, independent of a proper appreciation of their general causation, are attended with only temporary relief. It is only through a careful consideration of these cases from the gastrointestinal aspect, and the application of the proper therapeutic treatment along these lines, that a permanent resolution can be hoped for and maintained. How often are the middle, and even the inferior, turbinates more or less sacrificed in the attempts to relieve these conditions, when the result could have been more certainly, rationally, and, for the well being of the patient, more satisfactorily accomplished by properly applied therapeutic measures.

Another series of cases which claim our attention and that are provocative of disturbance within the upper air tract are those produced by errors in diet. It has been my observation that investigation of the diet of patients will often lead us to ascertain the cause of many cases of turgescence of the turbinates and various reflex disturbances in the oral and nasopharynx. In many such cases we will find the patients are partaking too largely of carbohydrates and sugar. Such patients are usually overfed, improperly clad, and do not take the requisite amount of physical exercise. The proper regulation of the diet and due regard to the habits of living will aid us very materially in bringing about restoration to the normal in this class of cases. Besides these cardinal general conditions cited, there no doubt occur to many of you others that could be brought forward wherein the chemistry of food, the biochemic action of various secretions, and the development of toxins within the body excite these deleterious actions directly or indirectly upon what is—for want of a better term —designated the vasomotor system of the respiratory tract.

It is only my intention in this short paper, in a general way, to aid in impressing again the important fact that there are many disturbances of the general system which influence the upper air tract from a pathologic point of view, and the relationship of these to the disturbances noted must be thoroughly weighed before any operative measure is resorted to; otherwise, disappointment may not only be keenly felt by the patient, but also by the operator, as the result of illmeasured intranasal operative efforts.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of April 11, 1913.

Paper: The Modus Operandi of Galvanic Nystagmus.*

By Lester Mead Hubby, M. D.

DISCUSSION.

Dr. Phillips said that he had had the pleasure of reading Dr. Hubby's paper, and had already congratulated him on the very effective work done in presenting a clear and lucid explanation of the action of galvanic irritation upon the semicircular canals. The diseases of the labyrinth are of great interest to all otologists, and it was necessary only to look over the programs of the section for the past year to note how much the minds of the members are turning to this topic, and to see how much has been accomplished in this line. Notwithstanding all this, we are still very much in the dark regarding the practical application of the knowledge possessed, which is necessarily still incomplete. The work which is being done, however, and the various papers which are being presented are helping to settle many questions. Dr. Phillips said that he had not himself given this phase of the subject the time and attention which Dr. Hubby had devoted to it, and therefore hardly felt competent to discuss it. Dr. Phillips said that Dr. Hubby had done most of this work in his service, and expressed the hope that other of the younger men would continue work along this line.

Dr. Isidore Friesner said that all the members of the section should be grateful to Dr. Hubby for his excellent paper. There are no other tests of ear conditions that are fraught with so much of discomfort to the physician as well as to the patient, and the utmost patience on the part of both is required. The results of galvanic tests are so surprising in their lack of uniformity that it seems impossible to evolve a classical picture. In suppurative labyrinthine disease the galvanic test gives us little or no information. We know from the experiments of Marx and from the examinations of Neumann and Ruttin that there is a reaction to galvanism long after the labyrinth is destroyed. He was surprised in noting Dr. Hubby's work to observe the effect of the galvanic current upon the auditory apparatus, at times increasing and again abolishing tinnitus. That seems to be an exceedingly fruitful field of study. There are many socalled cases of chronic catarrhal otitis where, in all probability, we are dealing primarily with neuritides and secondarily with atrophic changes in the end organs, and since observing Dr. Hubby's work he has wondered whether the galvanic test would not offer some help in differentiating these conditions.

---

*See page 638.

Dr. Ballin said that the subject of galvanic nystagmus as presented by Dr. Hubby was of great interest, but it is to be regretted that the consideration of a subject like this leaves us still very much in the dark, for all such papers are necessarily based upon theory and not upon practical experiments.

One of the most carefully prepared papers on this subject that he had seen was published by Brünings, dealing with the cataphoresis theory, and that the flow of the stream of endolymph is affected by the application of the anode and cathode to the mastoid region. You get a flow of the endolymph in one direction upon the application of the cathode, and the reverse when the anode is applied. This theory does not seem to be generally recognized, however. Marx of Heidelberg carried on a series of experiments on cats. In some of the animals he filled the labyrinth with lead, while in others he destroyed the labyrinth entirely. Upon applying the galvanic current he found that there was still a reaction in the form of a perceptible nystagmus. These tests tend to show that the galvanic nystagmus is due to irritation of the nerve endings, and, as Dr. Hubby says, to the nerve endings in the cristæ. The nystagmus produced by the caloric and rotatory tests are, however, the most reliable. It is found, however, that when the labyrinthine structures were destroyed by pus, etc., a galvanic reaction could still be obtained. According to Neumann, when he did not get this, he claimed that its absence was due to a retrolabyrinthine condition, and that he could thereby detect whether the lesion was located in the retrolabyrinthine or in the labyrinth itself. Of course, this is all theory, but Dr. Hubby's paper had certainly awakened thought for further investigation.

Dr. McCoy also congratulated Dr. Hubby upon his interesting paper. Such papers have both a theoretical and a practical value. We cannot write or talk too much about the labyrinth at present, for all the knowledge concerning it seems to be confined to the otologists. It is surprising to note how very little most general physicians seem to know about such conditions. He had recently been called in consultation with a physician whose opinion he values highly on most topics. This physician had asked him to open the ear drum of a woman patient, which he did. The physician said that he would look after the case, and five days later he stated to Dr. McCoy that this patient had tried to get up in the middle of the night, and was seized with a vertigo, and felt as if she were being turned about; she returned to bed and waited in this condition till morning for the physician, and he said she had an attack of hysteria. She was having some trouble with her husband. He treated her, and then asked Dr. McCoy to see her two days later. She then had quite a marked spontaneous nystagmus, and yet this physician had no conception of what was going on in the internal ear.

We cannot read or publish too much about the labyrinth, for it makes the subject more widely known and calls attention to it. Dr. McCoy said that he had hoped that Dr. Hubby would make some suggestions as to how to make galvanism more practicable. Up to the present time it has been more or less discarded because of the dictum of Neumann concerning it. The only improvement is that we may differentiate it by the fact that on the side on which the labyrinth is affected we have to use a stronger current to produce nystagmus.

Dr. Hubby said that galvanism makes the nerve more irritable, and in this way we may be able to bring out a spontaneous nystagmus; sometimes we think we have it but we are not sure. The theory of Bárány and that presented by Dr. Hubby are very interesting. Dr. McCoy said that he would be inclined to accept the view presented by Dr. Hubby. He thought it more logical than the view of either Bárány or Brüning. It seems to cover the field thoroughly and practically.

Dr. Fowler said that it was practically impossible, even for one who had looked into the subject quite carefully, to follow the spoken argument of a paper like Dr. Hubby's. The facts and the references to the labyrinth come with such rapidity that one cannot keep in mind exactly what the speaker is saying, and therefore it would be useless to attempt to discuss many of the points which suggest themselves, but by making a diagram upon the blackboard he thought he would be able to present one or two more points simply and clearly.

Dr. Hubby had spoken of the impossibility of electrically stimulating the semicircular canals individually. The same difficulty enters into every test method that we have today. Any fluid current in one must necessarily cause a current in another canal, either by pressure or siphonage, the latter especially in the two vertical canals, on account of their joining. A like way with the horizontal canals, etc.; the disturbance may not cause an actual current, but a tendency to change in the movement of the current in some degree. If we bear in mind the exact relationship of the orifices of the canals to each other, light will be thrown upon these tests and explain why they are not more accurate. The wonderful thing is that they are as accurate as they have been. He had made measurements on the actual bones of all of these parts, and these explain certain results obtained in practice from rotation, but it would be impossible for the members to follow the data off hand. The hydraulic interrelation of these canals should also be considered, although it may be that is not the important factor in their action. The placing of the maculæ of the canals is very suggestive. It can be easily seen that they may be affected by pressures in or of the endolymph, due to the centrifuging or flow of this fluid, whereas the maculæ of the utricle and saccule are so placed that they may be influenced by the faintest changes of pressures, but not by fluid flows. It may be that it is a combination of the endolymph flow in the canals and the pressure in the larger sacs which determines the labyrinthine influences.

Dr. George E. Davis agreed with Dr. McCoy, that the whole subject was difficult and not well understood by the general practitioners, indeed not by all specialists.

Dr. Hubby's explanation of the mechanism of the reflex of galvanic nystagmus was most interesting if not entirely convincing. It must be borne in mind that nystagmus is a reflex produced by some stimulation or irritation in various ways—mechanically, chemically and electrically. The different tests employed to elicit this impulse may be explained on different lines.

(1) Mechanically, by the stress, pressure or momentum of the endolymph current produced; (a) by rotation; (b) by air pressure or rarification, if a fistula be present; (c) calorically by hot or cold water.

(2) Chemically, by the application of irritants directly to the end organs of the static labyrinth.

(3) Electrically, by the application of the galvanic current.

Different theories have been offered in explanation of the galvanic as well as the other reflexes. Neumann claims that the mechanism of the galvanic reflex and the caloric reflex is more or less similar. Heat and the cathode or negative electrode have a stimulating effect on the end organs of the labyrinth, whereas cold and the anode or positive electrode have a sedative effect.

Another theory of the galvanic test is that it establishes an endolymph movement by kataphoresis and thus produces nystagmus mechanically, as in the rotation and caloric tests. However, it does not seem to him possible to explain the galvanic nystagmus reflex on the mechanical theory, for the reason that you cannot limit or circumscribe the effect of the galvanism on any particular crista, and, moreover, the current affects the nerve trunk as well as the nerve end organ, for it is well known that nystagmus may be elicited galvanically, even after the destruction of the labyrinth. Moreover, the galvanic test is not practical for two reasons: in the first place, it is not accurate, and in the second place, it is very painful to the patient.

Dr. Hubby, in closing the discussion, said that nearly all who had discussed his paper had spoken of the impracticable side of the galvanic tests, and he agreed with most of what had been said.

In many patients one can get no nystagmus whatever with the amount of current that can be used. Therefore the suggestion that it be used as a differential test between labyrinthine and retrolabyrinthine disease is of no value. A step in the right direction, although considered by many a plaything, is the nystagmograph of Buys of Brussels. He claims that with his little capsule which he puts over the eye he can detect nystagmus when using only a slight current. The apparatus, however, is cumbersome and expensive.

He had not spoken of the cochlear nerve tests, for he thought he had too much extraneous matter in the paper as it was.

Brüning's theory that galvanic stimulation of the vestibular nerve was produced by a movement of the endolymph was difficult to harmonize with the fact that with galvanism you can produce nystagmus whether the endolymph is there or whether the canals are all destroyed.

He did not wish to give the impression that he considered irritation of the crista in the rotational and caloric tests was due solely to pressure. But he thought it must be some combination of variations in pressure, torsion, and stretching of the hair cells.

He also thought that the combined hair cells of a crista were in a state of either hyperirritation, normal irritation, or hypoirritation.

Dr. Glogau asked if Dr. Hubby had made experiments on animals, to which Dr. Hubby replied in the negative. Dr. Glogau rejoined that he merely wished to know what experiments had enabled him to arrive at his theory.

Dr. Hubby replied that it rested on the work of himself and others in trying to find some explanation which would fit all the known facts. It was purely theorizing.

Dr. Glogau asked whether Dr. Hubby thought this was somewhat different from the others. He would like to have the theory tested in a series of experiments on either people or animals, and then try the conclusions.

Dr. Hubby replied that we know certain facts, and we try to find a theory to fit these facts. That was all that could be said. You can experiment on animals or on persons and get certain results, and then try to explain the results. Previous theories did not seem to explain the phenomenon, and he had tried to find something that would. Of course the exact mechanism of the stimulation of the hair cells in the crista in movements of the endolymph, etc., is still in doubt.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of May 12, 1913.

SYMPOSIUM ON RADIOGRAPHY OF THE MASTOID.

Exhibition of Radiograms Illustrating the Pathology of Mastoiditis.*

BY HENRY S. BIRKETT, M. D.

Radiographic Findings Illustrating the Anatomic Development of the Mastoid Bone. (Stereopticon Demonstration.)†

BY WM. H. STEWART, M. D.

Radiography as an Aid in the Diagnosis of Mastoid Disease.‡

BY FREDERICK M. LAW, M. D.

DISCUSSION.

DR. PHILLIPS expressed his regret that he had not been able to arrive sooner, and had seen only the last of the series of pictures presented by Dr. Law. He had seen Dr. Birkett's pictures at the International Otological Congress, and Dr. Birkett had recently asked him to express to the section his regrets that the change of dates had prevented him from the pleasure of being present, and furthermore, to say for him that he still held the views expressed at the International Congress in Boston on the subject of radiography in connection with mastoiditis—that while valuable as a diagnostic aid, he does not allow his clinical judgment to be biased by it.

Dr. Phillips said that he was familiar with most of Dr. Law's pictures. The one showing the septic condition of the lateral sinus was the only one which had been shown at the International Congress in Boston, and, so far as he knew, it was the only one of the kind in existence—i. e., showing involvement of the lateral sinus. He understood that the findings showed that it was really filled with pus and not a clot. It was very largely pus, if he remembered correctly.

---

*See page 649. †See page 677. ‡See page 635.

Two or three points in connection with radiography as an aid to diagnosis in mastoiditis are worthy of emphasis. First, the comparison of the plates. Having the two mastoid processes taken at the same time, with an equal exposure. Thus the same relative positions are given, and it is by the comparison of the two plates that the facts desired are brought out.

It is an anatomic fact that the mastoid processes are almost invariably symmetrical. The number, size, and extent of the zygomatic cells are the same, and if there is a large tip on one side, a similar one exists on the other. There are few exceptions to this rule. When one understands the interpretation of these plates as Dr. Law does, and as do some others who are not specialists in such work, the radiographs are an aid in confirming the diagnosis.

Another point is that it usually shows the position of the lateral sinus, and the operator thus has a fairly intelligent idea of the location beforehand.

A third advantage is that there are many times when the otologist feels that a certain case should be submitted to operation, but the family and sometimes the attending physician are averse to it. They are inclined to say: "No: I have seen cases just as bad get well." If, however, you have a radiograph showing clearly the difference between the two sides, to show to the physician or some member of the family, it is most convincing.

Another point which should go on record in discussions in regard to radiography of the mastoid process is that one should be able to make a diagnosis without it, and that we should not permit ourselves to rely upon a radiogram for a diagnosis of an acute purulent involvement of the mastoid; but it is of value as confirmatory evidence, and as such we should give it a place in modern otology. That is the view which Dr. Birkett also holds.

Dr. Freudenthal said that after hearing the interesting papers which had been presented and seeing the excellent pictures which had been shown, he felt he had little to add, especially as it had been only a comparatively short time since he had been having radiographic pictures made in mastoid affections. He had at home a half dozen pictures which he had intended to bring to the meeting, but seeing the great number of prominent radiologists on the program he had decided not to do so. He could only judge of their value in mastoid affections when compared with accessory sinus affections. While he has several hundred pictures of accessory sinuses, and a much smaller number of the other affections, and while he would not like to operate in many cases of frontal sinus affections without the aid of a good radiographic picture, he agreed with Dr. Birkett and Dr. Phillips that in the majority of cases of mastoid disease the clinical symptoms are so definite that we can very well get along without the aid of a picture. Perhaps, however, after using these pictures for a few years, he may have the same opinion in regard to pictures of the mastoid that he now holds in regard to pictures of the accessory sinuses.

Dr. Hirsch said that he thought credit should be given to the roentgenologist, because he has contributed certain additional data which will assist the otologists in determining both the indication and plan for the operation. Though this work is now almost entirely in the hands of the roentgenologist, the time is not far distant when the clinician, having been taught by the roentgenologist both the technic and interpretation, will himself employ this method of examination as he uses other diagnostic aids. The new tendency is visible in other lines of work in which roentgenology has contributed its quota of diagnostic data.

Dr. Judson Quimby said it seemed to him that the otologist is so able to make a clear-cut finding in regard to the mastoid, that there is very little left for the roentgenologist to do but to confirm his diagnosis. The clinical signs and symptoms of mastoid disease are comparatively clear-cut; whereas the findings in the deeper parts of the nasal sinuses, the ethmoids and sphenoids, are quite difficult. He had found difficulty in convincing otologists that much aid was to be obtained from the roentgenologist. All that he would claim on the mastoid would be to confirm the findings. He rather challenged Dr. Hirsch's statement that he would expect otologists to take up this work for themselves, for it requires a great deal of time, and no busy otologist could give it sufficient attention to do it well. Dr. Stewart's paper had demonstrated that something more was required than taking a couple of exposures to make a satisfactory mastoid examination. The mastoid should be studied from several different axes. One must have the two sides. If you get the two sides in a normal subject, you can possibly make a comparison, but it is rather difficult. The lateral sinus is especially difficult on account of its variation in the two sides. The right lateral sinus is more spread out and not so clear-cut as the left one. If one will notice a large number of plates, the difference in the two sinuses will be observed. His own technic is about the same as that of Dr. Sidney Lange, of Cincinnati, who has done a great amount of valuable work.

Dr. Sinclair Tousey said that he was probably the first one who suggested the practicability of making X-ray examinations in cases of mastoid disease, having suggested it to Dr. Whiting some nine years ago, but that Dr. Whiting had replied that it was possible to make the diagnosis without it.

The pictures shown by Dr. Law, taken with the patient lying on the back, the occiput lying on the plate, were excellent, but that the same position could be modified by turning the patient's head somewhat to the right for the first plate, and then making a second plate with the head turned somewhat to the left, showing the mastoid cells in their normal position.

The following are some memoranda of the author's methods: There are three principal methods which have proven most useful in his practice. One gives a simultaneous and practically unobstructed view of both mastoid processes. The plate is a large one and is in front of the face, while the tube is in the median line behind the head, at about the level of the occipital protuberance. The mastoid processes are at such a distance from the plate that in order to secure a clear image the X-ray tube should be at a considerable distance from the head, and a diaphragm should be used to exclude secondary rays. There is a very great advantage in having a picture of both sides taken under identical conditions.

The second method is one devised by himself, and has not previously been published. The two sides are radiographed separately, taking great pains to secure symmetrical positions. The plate is at the affected side of the head, which is bowed, and the face turned away from that side: the X-ray comes from behind and just above the opposite shoulder, passes behind and below the opposite mastoid process, and produces an unobstructed image of the mastoid process, which is almost in contact with the plate. This is to be compared with an identical one made of the other mastoid process.

The third method is one described in his book ("Medical Electricity and Roentgen Rays," W. B. Saunders Co., 1910), in which the X-ray shines through the head horizontally, along a line one inch above the external auditory meatus. This gives a view of the mastoid cells at the level of the tympanum and also a view of the labyrinth.

There are certain general principles which are of prime importance. One is in regard to the use of the fluoroscope. The voice of the tempter tells us that we may safely see enough to enable us to properly adjust the patient and the plate and the X-ray tube. But it is just this kind of work which has caused the suffering and death of so many of the best roentgenologists in every part of the world. It should be absolutely prohibited as dangerous to operator and patient.

A radiograph through any thick portion of the body is always very much better if a diaphragm is employed to cut off secondary rays. And the diaphragm should be made of material opaque to the X-ray but a nonconductor of electricity, so that it may be placed in contact with the wall of the X-ray tube and give a wide angle of radiation.

Intensifying screens may now be had which show practically no grain in the picture and which reduce the time of exposure to one-tenth. I should recommend their routine use for these cases. The same effect is produced by the modern transformer or by the still more recent unfluctuating converter in a fraction of a second as by the old induction coil in a minute or so. And where it may be desirable to make several pictures, the element of X-ray dosage becomes important.

A recent improvement in X-ray generators is the result of a suggestion contained in his book. It is in effect a dynamo producing a direct current of uniform high voltage and an approximately uniform wave-length of X-ray. Regulating the apparatus for a radiograph through the head causes the production to a less extent than ever before of those X-rays which would be arrested and absorbed by the superficial tissues and have a tendency to burn without taking any possible part in the production of the radiograph, because they never reach the plate. This is a great element of safety in X-ray examinations and also in the treatment of deep seated lesions by these rays.

Dr. Seymour Oppenheimer said that personally he did not think radiography would be of much value in the great majority of cases in the interpretation of disease of the mastoid. In some obscure cases it might be of great value, but they were the exception. Clinical evidence must make our diagnosis and not the radiogram.

One point of great value mentioned by Dr. Law was the recognition of anatomic details. Most operators have at times met with misfortune when chiseling a little too energetically and struck the sigmoid sinus unexpectedly; even when using great caution in keeping close to the posterior bony canal wall, he had at times been mortified to find the sigmoid sinus directly beneath, in a far anterior position, occupying a very superficial position. A radiograph taken as a routine measure would give exact knowledge of the location of the sinus. It would seem to be more valuable for outlining the anatomic details in chronic rather than in the acute type of cases. They would also be of value if a series of radiograms were taken in a case where there is some doubt from the external or subjective manifestations. If the radiograms were taken on a given day and show a slight blurring of the structures, and then another taken several days later showed further blurring, it would be suggestive of an extension of the purulent process. Such an instance represents the value of the radiogram, but not as a general means of establishing a diagnosis.

Dr. Felix Cohn said that he wished to emphasize the point made by Dr. Oppenheimer—that the value of the radiogram at present is not in enabling one to make a diagnosis of mastoiditis, but in giving information in regard to the anatomic details, as we get it in the frontal sinuses, so that we may know beforehand the size and configuration of the sinuses. The point of emphasis is the knowledge of the anatomy before commencing the operation.

Dr. Hubby said that he would like to call attention to one point in the examination of children—especially sick and nervous children. If you examine first one side of the head and then the other, and simply look at the mastoid process without considering the whole picture, you may get an entirely wrong idea of the conditions, for the child is very liable to move, especially when the diseased side is against the table and plate. If you should find the mastoid on that side blurred and the other side clear, you may come to the conclusion that it is diseased, when the blurring is simply due to a faulty picture. In one or two of the pictures shown by Dr. Stewart it seemed possible that he might have made a misinterpretation of some of the plates. In one case in which he said the character of the cells of the two sides were dissimilar, one plate was blurred while the other side was clear. That always has to be taken under consideration in cases of children. For that reason it would seem advisable to have a single plate taken from the rear, giving a bilateral view, as suggested by Dr. Stewart, as well as the usual side views.

Dr. Stewart is ceratinly to be congratulated on getting such excellent plates in such difficult subjects as young children.

Dr. Van Wagenen said that the first operative case shown by Dr. Law was that of a patient who had been referred by him to Dr. Law with an acute mastoiditis. Immediate operation was contemplated, but he was persuaded to wait on account of the skiagraph. The first plate showed purulent involvement of the aditus. The tip was clear, as were also the cells, whose partitions were sharply defined. The plan of treatment was changed entirely, and operation was delayed for four days. The symptoms then became pronounced. He went in, opened the mastoid, and found the tip and cells free. But a pocket of pus over the sinus compelled him to expose the dura before he got it entirely eradicated. If he had followed his own desire in the matter and operated early, instead of placing so much reliance on the skiagraph, he would have saved at least forty-eight hours and avoided an exposure of the dura.

Dr. Voislawsky said that the plates were valuable in mastoid operations on certain cases where speed was necessary, as in diabetics or cardiacs. If the exact location of the sinuses were known, several minutes might be saved in the operation, to say nothing of the element of safety to the patient.

Dr. Phillips said that he wished to emphasize one fact before the discussion was closed—that there is only one kind of radiograph which is of any value in the interpretation of mastoid cases, namely, a good radiograph. Many of them are poor, and unless the radiograph is a good one and can be contrasted with an equally good one of the other side, it is not well to rely upon it. At the Manhattan Eye, Ear and Throat Hospital they were extremely fortunate in having a roentgenologist in Dr. Law upon whose work they could rely. Furthermore, he would give full credit to Dr. Caldwell, who was the pioneer in making satisfactory radiographs of the cranium.

Dr. Stewart said that he had little further to add, as most of the discussion did not refer to the development of the mastoid cells. He would like to emphasize, however, that team work was absolutely essential for successful use of the X-ray as an aid in the diagnosis of lesions of the mastoid. The most important value of the radiographic examination was to give accurate anatomic relations in contemplated operative interference. He had stated that it was absolutely necessary to have radiographs of both sides and of about the same density in order to make accurate comparative studies. The radiographic studies that he showed were taken from a number of normal patients in the wards of one of the hospitals with which he is connected. They were taken at random, and the radiographic findings were not selected. He said this in order to show that it is not unusual to find pneumatic cells in children of two, four and five years of age.

He could not agree with Dr. Hirsch, that this work was very simple in its procedure. He had been in busy offices of a number of otologists, and it hardly seemed possible that they would care to take up the work of examining mastoids in addition to their numerous duties.

He expressed regret that Dr. Tousey had not brought some of his radiographs to the meeting, that he might demonstrate the advantages of the methods he advocated.

Dr. Law said that the best illustration he could give of the way in which otologists are taking up radiography was the fact that whereas two years ago at the Manhattan Eye, Ear and Throat Hospital they made plates of possibly one mastoid in two or three months, they now make two or three a day—not so much for diagnostic purposes as for a registration of the anatomy and the location of the lateral sinus. It can be located absolutely and accurately—not as shown in the pictures presented tonight, as they showed the whole structure, taken from an angle, but by taking pictures directly through from one meatus to another or by stereoscopic plates.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of February 18, 1913.

THE PRESIDENT, DR. J. GORDON WILSON, IN THE CHAIR.

Demonstration of Cases.

DR. G. W. BOOT presented the case of Mr. J. McN., aged fifty-four years, janitor by occupation, who, about the middle of December, fell on the ice, striking his head in the region of the right occiput and mastoid. Blood came from the right external auditory canal at this time, and the patient became quite dizzy—so much so that he could hardly keep on the sidewalk when he walked home. He had been more or less dizzy ever since, although the dizziness was gradually passing off. Two days after the accident he had diplopia. This had also passed away. There was at present no spontaneous nystagmus and no unsteadiness in station. Both ears responded to the caloric test. Drum membrane on the right side was thickened; on the left side was about normal. Hearing tests: Bone conduction shortened. Weber (C) to the right. Rinné: right, plus; left, plus. Low limit: right, heard 32; left, heard 32. High limit: right, 9,000, d. v.; left, 19,000, d. v. The upper half of the optic discs was redder than the lower half, and the margin was rather indistinct, but choked disc was not preesnt. Blood pressure, 140.

Exhibition of Specimens.

DR. NORVAL H. PIERCE had very little to say regarding the specimens presented. They were nearly all marked, and were simply specimens of adenoids and tonsils. There was one specimen of adenoma of the larynx, and another of a tumor of the nose, which grew from the region of the sphenoid bone—rather large—in a woman advanced in years, which very closely resembled carcinoma, but inasmuch as there was no recurrence and removal was not especially thorough, the diagnosis of carcinoma could not be sustained. He expected to find in the collection, which was picked up very hurriedly, a very beautiful specimen of adenoid tissue showing the distribution of the blood vessels, giving a very good idea of the way adenoids undergo involution. The blood vessels supplying adenoids all converge toward a rather limited space, and it is supposed that the connective tissue at a certain time constricts around the blood vessels at the base and thus deprives the rest of the body of the adenoid of its nutrition. He has not found it there, among the specimens. But, as he said, the specimens all speak for themselves.

Pin-Swallowing.

Dr. S. C. Friedberg reported a case of pin-swallowing in a girl of thirteen years, with hysteria. Esophagoscopy was performed.

Relation of Mucoperichondrium and Periosteum of the Nasal Septum.

Dr. Otis H. Maclay said that the specimens showed the relation of the mucoperichondrium and periosteum to each other and to the bony portions. Certain anomalous conditions are present. In one specimen the perpendicular plate of ethmoid occupied almost the entire anterior portion of the septum, leaving only the extreme lower anterior area for a small piece of cartilage.

Discussion of Cases and Specimens.

Dr. Joseph C. Beck said that he had seen the case presented by Dr. Friedberg at the County Hospital, when she first came to him. He got the history that she had swallowed pins. He had a picture made the same evening. In examining the patient he could find nothing in the way of a pin. The girl was typically hysterical, and on questioning the mother it was found that this girl had had a fright by a little boy when she had some pins in her mouth. This fact was not stated by Dr. Friedberg. Possibly this was a factor.

The speaker had a similar case a year ago, which was still more interesting. A girl said she inhaled a pin. He could find nothing, and so had an X-ray picture taken. In the picture a pin showed about an inch away from the soft structures of the back, over the clavicle. On pressing the girl for a definite answer, she finally said that she knew that it would show in the plate, so kept the pin in her hand and placed it on the plate.

Regarding Dr. Boot's case, he thought there were many things in a case like this that ought to be developed; for instance, the blood pressure, urinalysis, etc., in reference to an arteriosclerosis, which possibly would explain some of these conditions, aside from a pathologic process at the base of the skull. With a pathologic process it seemed to him there should have been more inflammatory conditions.

Dr. George E. Shambaugh called attention to the fact that one of the most common types of malingering is the claim of defective hearing due to accident. It is a common belief that one may assert that he has lost his hearing through an accident and that it is impossible for a physician to discover malingering. He sees a great many railroad cases of this type. As a matter of fact, one usually has very little difficulty in discovering malingering when an injury to the ear is claimed. If there has been an actual injury to the internal ear the symptoms of deafness and tinnitus and vertigo are so characteristic that even when the patient has heard about these symptoms and has not experienced them, he is unable to tell us exactly how they occur. For example, he examined a case a day or two ago. A young man claimed to have been struck by an auto truck and knocked from the steps of a street car. He walked home without any difficulty, and claimed that that night he had a very bad earache, which had kept up ever since. Examination showed the drum membranes were normal. He also stated that he was very dizzy; that he was unable to lie down, and had to get up and walk around. In making the Weber test he insisted that he was unable to hear the tuning fork if he put his finger in the well ear, but could hear distinctly with the ear left open. A history such as this showed clearly the confusion a patient usually has in trying to assume symptoms which he does not experience.

Dr. William L. Ballenger recently had a case of a man who, when riding in an automobile, struck a telephone pole and was unconscious for some days afterwards; he was very giddy and nauseated, and so forth, and he presumed had nystagmus. No observations, however, were made in this regard. Dr. Ballenger saw the man for the first time three months after the injury, and up to that time he had had some difficulty in walking. All these were phenomena of reaction movements, and the X-ray plate showed a fracture running from one ridge up over the vertex, almost, and around to the opposite supraorbital ridge. It also showed fracture in the base of the skull. Otherwise, he had total loss of hearing in this ear. But apart from this he had made practically a complete recovery. Several X-ray pictures were made of his head at various angles, in order to see if there was any depression of the bone, but there was nothing—simply the fracture without any depression—which accounted for his good condition. The only damage apparently now is his total loss of hearing in the affected ear; the other ear is entirely normal. When the speaker saw him he still had a blood clot in the drumhead of the unaffected ear, but in the affected ear the drumhead was normal. He could not explain this, but simply reported it as part of the case. He thought Dr. Boot's case was one in line. He did not know whether Dr. Boot had plates made or not, but in his case the X-ray showed the fracture very clearly.

Dr. J. Holinger said that of course all were agreed that Dr. Boot's was most probably a case of fracture of the base of the skull. There is a work published in which the writer has given a large compilation of these fractures of the skull and found that the labyrinth and the middle ear are places of predilection for fracture lines. The symptoms must vary according to the parts involved. He had an opportunity of seeing quite a number of these cases in his service at the Alexian Brothers' Hospital. A few years ago there was something like an epidemic of hold-ups by sand-bagging. The hold-up man came upon his victim from behind and hit him with a sand-bag over the ear; the victim would fall down and be unconscious for a longer or shorter period of time. Dr. Holinger saw five or six cases of fracture in cases of this kind, and a number of them were very serious as to their consequences. He observed one man for nine months because of the continued dizziness, which absolutely incapacitated him for work. The moment he tried to stoop he would fall down and, being a laborer, of course that made it impossible for him to find any kind of work. He finally disappeared. No logical treatment was indicated.

Another case, which he had reported to the society before, was that of a man falling from a height of eighteen feet on his head, with consequent hemorrhage from the left ear and unconsciousness, which latter lasted for two or three weeks. Every two or three days he would wake up for a short while and fight violently in bed, trying to take hold of something or somebody. After a little while he would relapse into unconsciousness. He recovered, but there was no hearing in that ear. Dr. Holinger asked him why he fought so violently when he became conscious, and he said when he woke up he would see the room and everything in it normally for a short moment. Soon everything began to turn faster and faster, and he thought that he was falling, and with his utmost power he held himself on whatever he could grasp. The dizziness became worse until he lost consciousness.

The differential diagnosis between fracture and malingering is not very difficult, if you remember the function of the semicircular canals. Often the hearing tests will be of service.

Dr. Alfred Lewy does not think that the diagnosis in an injury case is always so plain. He remembered the case of a man who received a severe head injury in a railway collision. He was seen at his residence about a week afterward. Aside from general nervous symptoms he complained of pain in the ear, noises, and some vertigo. The drum membrane had the appearance of an acute otitis media, and paracentesis was performed. A week afterward the patient reported at Dr. Lewy's office, and tuning-fork tests showed every evidence of a pronounced nerve deafness. Dr. Shambaugh also saw this case and diagnosed nerve deafness. There had been no hemorrhage from the ear, and no organic lesion of the brain was diagnosed. The man was partially invalided for the greater part of two years as the result of his injury.

Dr. J. R. Fletcher thought one thing should be mentioned regarding Dr. Holinger's nine months' case. It seems to him that in a case of this kind, if the labyrinth had not been totally destroyed, the treatment should consist in destroying it. This man was a laborer and could not work because of the disturbance caused by the partially destroyed labyrinth, and it seemed to him that the treatment above suggested would be the best course to pursue.

Dr. J. Holinger did not know whether he would call the treatment suggested by Dr. Fletcher absolutely good surgery, because we do not know how the fracture lines run, and by operating we may cause a fatal hemorrhage, either subdural or intradural, even at some distance from the place of operation, and unknown to us. The more postmortems one sees of such cases, the more one will be impressed that absolute rest is indicated.

Paper: Topography of the Tympanic Cavity.

By John A. Cavanaugh, M. D.

DISCUSSION.

Dr. George E. Shambaugh complimented Dr. Cavanaugh on his beautiful anatomic preparations, and emphasized that everyone who aspired to become a thorough specialist in otology should undertake to work out for himself in this way the anatomy of this region. Anatomy cannot be learned from textbooks or atlases; one must handle the actual specimens in order to get that knowledge which will be of real assistance when working clinically with the ear, and the best way is to make one's own specimens, which will illustrate the various points. In glancing at a specimen of the temporal bone one might suppose that the anatomy of the tympanic cavity could be described in a very few words, but after listening to Dr. Cavanaugh's excellent paper on this subject, one can realize that he has to deal with a very complex anatomic structure. Many of these anatomic facts may at first thought appear to have very little practical interest, but, as a matter of fact, every point in the anatomy of this part has a clinical bearing.

Dr. Shambaugh was interested in the preparation which Dr. Cavanaugh exhibited, showing the aberrant pneumatic spaces extending forward in the petrous portion of the temporal bone. Dr. Shambaugh has a number of specimens of this sort. Infection in these cells cannot be reached by our usual operations.

Dr. Shambaugh called attention to the socalled tympanic cells which develop in the floor of the tympanum. He has several preparations in which these cells extend fully half an inch from the tympanic cavity. He called attention to the constant pocket just back of the promontory, to which the term sinus tympanicus has been applied.

There was one feature in the anatomy of the tympanum which was not brought out in the part of the paper which Dr. Cavanaugh read that seems to have a very important clinical bearing. This is the duplication of folds of mucous membrane in the cavum tympani. These folds are normally found extending between the fan-shaped ligament arranged horizontally around the neck of the hammer. They frequently separate almost completely the cavum tympani proper from that part of the recessus epitympanicus, to which the term attic is usually applied. Other folds are frequently found connecting the cruræ of the stapes with the niche of the oval window. Another fold is often found connecting the long crus of the incus with the posterior wall of the tympanum. Dr. Shambaugh has a specimen where a fold of this sort divides the antrum, the aditus and the attic into two compartments. These folds of mucous membrane frequently form the basis for those thickened bands that develop in the chronic adhesive forms of middle ear disease. Normally, the folds are made up of two layers of epithelium, between which are located a few capillary vessels. In those individuals where these folds are well developed, either a prolonged middle ear inflammation or repeated attacks of acute inflammation lead sooner or later to the development of connective tissue which produces a marked stiffening in the conducting mechanism. In other individuals, where these folds of mucous membrane are not well developed, there is much less likelihood of permanent disturbances of the hearing resulting from middle ear infection.

Dr. A. H. Andrews heard Dr. Cavanaugh mention the spiculum of bone which frequently extends from the apex of the pyramid across to the inner wall. He has frequently observed that in his studies, but so far has not seen it mentioned in any textbooks on anatomy, and thinks that if it is not mentioned, Dr. Cavanaugh should be given credit for calling attention to it.

Another thing which he thinks should receive more attention is the relation of the facial nerve in its downward course to the posterior wall of the tympanic cavity. He has found many specimens in which the plate of bone lying between was as thin as bone could well be. Randall mentions two millimeters, but it is frequently very much thinner.

Dr. Shambaugh mentioned the necessity of handling the specimens. The thing that has been of great value to him in the study of the temporal bone has been the making of clay models of the bone, especially clay models of the labyrinth. He thought he had a pretty fair knowledge of the gross anatomy of the labyrinth until he undertook to make clay models from Wood's model cast, and he found he knew very little about it until after he had made a number of models, and he is constantly finding things as he has occasion to work with the clay that had previously escaped him. He thinks that it is an excellent plan to make models of different parts of the middle ear, and the internal as well. He has noticed a great variation in the attachment of the tendon of the stapedius muscle to the stapes. Most of the textbooks say that it is attached to the head of the stapes. He has found it attached to the long process of the incus, but more frequently to the neck of the stapes or its posterior crus, than to the head of the stapes. He thinks much is to be accomplished by careful study; indeed, it is absolutely essential, if we are going to do efficient ear work, that we put a great deal of time on the anatomy of the ear.

Dr. George W. Boot has been under a different impression than Dr. Cavanaugh, for in his specimens of temporal bones at birth the annulus is not separate from the squama.

With reference to dehiscences of the floor of the tympanum, large dehiscences which would permit the jugular bulb to be seen through the membrana tympani are certainly not very frequent. In ten years' work at the Central Free Dispensary he had seen but one case in which the diagnosis had been made.

One point about the anatomy of the tympanic cavity was not mentioned by Dr. Cavanaugh, and this is the distance from the manubrium to the promontory. Most of the textbooks are erroneous in that they represent this distance much greater than it should be.

The cellular spaces mentioned by Dr. Cavanaugh as extending from the upper part of the tympanum into the upper wall of the canal are of considerable importance, for the burrowing of pus into these cells occasionally causes the formation of a fistulous opening into the canal some distance external to the membrana tympani.

Dr. Cavanaugh gave the thickness of the drum membrane as about 1/10 mm. It is remarkable to what extent this thin drum membrane can swell in suppurating conditions of the middle ear. It is not uncommon to see a drum membrane which is 2 or 3 mm. in thickness as a result of suppuration.

He hoped Dr. Cavanaugh would pardon him if he called his attention to two errors in nomenclature. The manubrium is not the long process of the malleus, and the antrum is not the mastoid antrum, but is the antrum tympanicum.

Regarding the supposed change in the position of the drum membrane, it seems to him this question could be quite easily settled by measuring the angle which the groove of the annulus tympanicus on one side forms with the groove of the annulus tympanicus on the other side in the newborn and in later life.

In this connection, he called attention to two or three specimens on the table, one showing an anomalous course of the canal of the chorda tympani. Another specimen showed the relation of the antrum to the tympanum at birth, at which time the aditus is almost directly above the tympanic cavity.

Dr. Joseph C. Beck was sorry that the essayist had skipped a large part of the paper. The reading of the same will be of great interest; also the illustrations.

Regarding dehiscence, he called attention to the work of McKernon in infants, particularly in the region of the jugular bulbs, which are very numerous, contrary to what Dr. Boot said.

Regarding the cellular spaces referred to, you will find in X-ray pictures cellular areas reaching from the tympanic cavity to the sphenoid as one continuation of cells. You must deetrmine whether these socalled cellular spaces are really cellular spaces in connection with the cavity lined with this epithelial mucous membrane, or are of diploeic bone. He has some histologic sections of this particular portion, and has found many of them are diploe.

Dr. Cavanaugh, in closing, wished to thank the members for discussing his paper. Owing to the lateness of the hour, he omitted reading the entire article, which would have covered the remarks of Drs. Shambaugh, Boot, Andrews and Beck, but he mentioned that when the article appeared in full it would be found to deal with their suggestions.

Regarding Dr. Boot's statement that a dehiscence is rare in the floor of the middle ear, Dr. Cavanaugh wished to say that he has seen that condition in a pathologic specimen.

As to the folds of the tympanic cavity, discussed by Dr. Shambaugh, reference was made to them in the paper, but omitted for lack of time.

Paper: International Otological Congress in Boston, 1912: (a) Otosclerosis; (b) Discussion of the Demonstrations of Professors Urban Pritchard and Denker; (c) Heath's Paper.

DR. J. HOLINGER said that the demonstration of microscopic specimens of Professor M. Pritchard, of London, and Professor Denker, of Halle, contradicted each other. Professor Pritchard said that birds have no basilar membrane in the labyrinth; Professor Denker showed very beautiful specimens of it. This finding is important for the theories of hearing. The same is true of specimens of Professor Siebenmann, who showed labyrinths of guinea pigs where the incus was removed from one middle ear, and the animal exposed to noises calculated to produce degeneration of Corti's organ. The cochlea where the incus was removed remained normal, the other one was degenerated. Both of these findings speak in favor of Helmholtz's theory of hearing, and against Zimmermann's.

Finally, attention was drawn to a demonstration of Professor Siebenmann of specimens and drawings of otosclerosis. The pathology of this disease was attacked in a book by Professor Manasse, and also the diagnosis as given by Bezold. The specimens counter-prove Professor Manasse's attacks.

DISCUSSION.

DR. J. GORDON WILSON said that he read Manasse differently from Dr. Holinger. He understood Manasse to say that otosclerosis is primarily a hereditary degeneration. If he remembered Manasse's pictures aright, they showed in the labyrinth capsule as the earliest sign a collection of what he called granulation tissue in close relation to an artery. Then followed absorption of the old bone and redeposit of new bone. Manasse did not believe it to be a specific disease of the stapediovestibular attachment, but of the labyrinth capsule. Subsequent to this come the changes in the nerves of the cochlea and the cells of ganglion spirale.

Of course, the whole question is still extremely obscure, but it is satisfactory to find two opposing views so energetically discussed. The outcome cannot but give a more accurate knowledge of the etiology and pathology of this obscure disease.

Dr. George W. Boot said, with reference to the presence or absence of a basilar membrane in birds, that he had cut sections from the lagena of the owl, bluejay, woodpecker and the common barnyard fowl, and that in these birds at least a basilar membrane was present.

Dr. Shambaugh called attention to one point which the specimens exhibited by Dr. Urban Pritchard and Dr. Denker brought out. Dr. Urban Pritchard has held the view that the cochlea in the bird's ear has but two scalæ—a scala tympani and a scala media—and that there is no Reissner's membrane. Dr. Denker holds the view that the scala vestibuli is present. The Reissner's membrane is quite a different structure in birds from what it is in mammals, but it is still there. Dr. Urban Pritchard showed specimens of the labyrinth taken from the ornithorhynchus, the duck-bill of Australia, which showed that the cochlea in this interesting animal possesses the characteristics of the bird's cochlea rather than those of the mammal.

Dr. Joseph C. Beck said that a great many of the cases operated on by Mr. Heath in Boston had required reoperation. Mr. Heath is an enthusiast and has created a semiradical operation and given some people a moment to think before operating every ear radically. So far as the speaker was concerned, he has not been very successful in the use of Heath's operation, except in children, and he thinks that the indications for the operation are rather the point that Mr. Heath leaves out, and the pathologic conditions that are found in the bone.

Dr. Beck is interested in the pathology of otosclerosis, and especially in the work of Gerber. Gerber, in his opinion, is the coming man. His differentiations coincide with those of

Gray in the study of otosclerosis, and also with the processes that are found in osteomalacia and rickets, and in some forms of degeneration as they are found in asthmatics in the upper respiratory tract. The speaker has been working along this line, and has had a good deal of correspondence with Dr. Gray in reference to this, and it is, in his opinion, a subject of great interest.

He believes that the standpoint of Manasse is worth while considering.

Dr. Holinger, in closing, said that the standpoint of Manasse certainly is worth while considering, otherwise we would ignore it. Every criticism has to be taken into account, but he thinks it is necessary to weigh the merits and demerits on both sides. The idea of spongifying being an inflammatory process leaves us absolutely in the air as to the clinical course. The word "inflammation" tries to explain the pathology, but no reason is given why it is more justified than "abnormal growth."

With reference to the question of inflammation: We have repeatedly heard it said that the identification of pathologic processes of the bone is one of the most difficult problems. Dr. Holinger has studied the work of Manasse quite recently, and has seen the areas described by Dr. Wilson, which are circumscribed with lines in the picture. But Professor Manasse does not say why he calls these areas inflammatory, or why they might not be due to abnormal growths.

Dr. Holinger states that Siebenmann has examined a great many young individuals, while Manasse has no patient below forty. Later on there are new formations, and just that circumscribed focus in the very old individual explains it.

Dr. Wilson said he did not think so.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of March 18, 1913.

DR. J. GORDON WILSON, PRESIDENT, IN THE CHAIR.

Stenosis of Right Auditory Meatus.

DR. C. H. LONG reported the case of Miss J. C., aged twenty-one years; lives at home. On September 13, 1912, she presented herself with pain and a discharge of the right ear. Family history negative. Personal history: Always healthy until the age of nine years, when she had the measles. From that time until her eighteenth year (1910), she suffered from phlyctenular keratitis, the corneal opacities becoming so opaque that her vision in both eyes was often as low as 15/200, even with the help of her correcting lenses.

The advice for the clearing out of the throat was not followed until April, 1912, when the phlyctenules reappeared.

In April, 1910, when the eye trouble had abated, an acute eczema appeared around the alæ, bridge of the nose, and side of the cheek. About September 5, 1912, an itching, burning sensation was felt in the right ear, followed by pain and a watery discharge. On the 13th he examined the ear, and, in view of her past history, was unable to make an exact diagnosis. The microscope showed only the presence of pneumococci. September 24th, the symptoms demonstrating a rather deep infection, with the possibility of pus formation, he made a free incision through the most sensitive part to the bone. This was done under gas anesthesia. No pus was found, but immediate relief from pain ensued. Cleansing with mild antiseptic solutions followed, but without much, if any, benefit. A thin, mucopurulent discharge came from the inner side of the narrowed opening of the external auditory meatus. It was impossible to view the drum membrane, so there was no way of telling the condition of the middle ear. The hearing was lowered, to be sure, but no more than we should expect with an occluded meatus. The lumen continued to become smaller, so that by January, 1913, three months from the beginning of the infection, the meatus scarcely allowed the passage of a small probe. He tried stretching with graduated sounds, then inserting different varieties of drain, sterile tin foil, moulded in the shape of a tube, rubber tubing, and gauze, but with unsuccessful results. He then used a slippery elm tent, cutting it down to fit the hole, taking care that the string was strong and properly tied, so there would be no danger of the swollen wood slipping behind the stricture. A second and larger tent followed the first. As a result of the dilatation, the drum membrane was inspected and found intact. In two or three days the meatus was nearly as small as before. It was at this stage that he presented her to this society.

On January 21st, under ether anesthesia, he performed the Körner flap operation. In a couple of weeks nearly all discharge had ceased and all wounds healed, with the exception of a spot external to the stricture in the meatus, where the flap did not cover the cut surface. This is gradually filling in, and he hopes soon to report a successful termination of a seemingly simple infection, but one that baffled all ordinary therapeutic measures. In this case he had to deal with an acquired occlusion of the canal, due to an infection seemingly deeper than the glands and hair follicles. Vaccines were not used in this case, owing to previous failures in former furuncular cases.

Referring to the flap operation, he should have met with greater success had he inserted a graft on the raw surface or else used the Jansen modification of Körner's operation.

Otosclerosis.

Dr. J. Holinger presented a series of specimens of otosclerosis presented to him by Prof. Siebenmann, of which some had been shown before. He had one new one which had two foci, one of which does not reach the surface at any place. From this it may be argued that under certain circumstances the process may start in the intermediary part of the bone, without touching the surface of the periosteum. This is an argument against the inflammatory nature of the process. He also showed one specimen of artificial boiler maker's deafness in a guinea pig, in which the incus was removed in one ear. Corti's organ in the operated ear was intact; in the non-operated one it was degenerated. Proof for Helmholtz's theory. He also showed one specimen of the labyrinth of a guinea pig which was exposed for fifty nights to a whistle with the clear sound of small "g." It shows circumscribed degeneration of Corti's organ.

Gross Lesions of the Middle Ear.

Dr. J. Gordon Wilson presented specimens showing gross lesions of the middle ear. They were obtained from patients in a lunatic asylum who showed marked hallucinations of hearing. It is interesting to note that in cases of aural hallucinations there are found a large percentage with marked ear disease.

Acute Mastoid Abscess and Temporosphenoidal Abscess.

Dr. Wilson also showed a young lad of twenty-one on whom he had operated three weeks ago for acute mastoid abscess and temporosphenoidal abscess on right side. The lad had before the operation severe pain in the right parietal region, which dated back to an accident four weeks before Dr. Wilson saw him. The X-ray pictures showed two fractures of the skull, one in the parietal and another in the squamous temporal. A full history will be published later.

Esophageal Bougie Fitted With a Wax Spindle-Shaped Enlargement for Dilatation of Cicatricial Strictures of the Esophagus.

Dr. Otto T. Freer showed an esophageal bougie fitted with a wax spindle-shaped enlargement for dilatation of cicatricial strictures of the esophagus. Dr. Freer was led to create the device by a case of stricture of the esophagus just above the cardia, which he had been keeping open for some years by monthly passage of the usual ivory olives, an especially made hard rubber olive, seven-eighths of an inch in diameter, completing the dilatation. Of late, however, the esophagus had begun to pouch above the stricture, so that the olives merely pushed down this pouch and failed to pass. A flexible gum elastic bougie, No. 20, however, entered the stricture readily enough. In order to accomplish dilatation with this bougie, Dr. Freer wound a spindle of dental floss silk upon it at the junction of its lower and middle third, and made the spindle solid by painting it with boiling hot dental moulding wax. The surface of the spindle was finally smoothed with a layer of still fluid but cooler wax, and the form of the spindle was finally shaped by scraping it with the edge of a knife. The upper end of the bougie was then cut off and a whalebone rod introduced as far as the spindle to stiffen the upper portion of the bougie. The spindle readily followed the flexible distal third of the bougie through the stricture and dilated it easily.

Dilating spindles of the kind described may be made of any desired size upon the regulation bougie. Dr. Freer regards these dilators as superior to distensible rubber bags, which are harder to pass than the ordinary olive.

DISCUSSION.

Dr. Joseph C. Beck said that Dr. Wilson's case had been exceedingly interesting to him. Regarding this socalled dead area—or the area in which diagnosis is very difficult—he had had a personal experience recently. His brother had succumbed to a tumor located in this region. That a tumor the size of a small apple can exist in this area without producing any symptoms is certainly a strange thing to him, and he is studying his brother's case from every point. This subject is of extreme interest to all rhinologists and otologists, because he believes it is in their sphere to assist in making the diagnosis. It is surprising that such an amount of pus could be obtained in this area without causing any symptoms other than pain.

Dr. Shambaugh referred to a case of brain abscess which had recently come under his observation. The chief complaint of the patient was severe onesided frontal pain of several weeks' duration. When first seen by Dr. Shambaugh there was a subnormal temperature, slow pulse and complaint of unbearable pain over the left eye. The patient stated that he had lost ten pounds in weight, and the clinical condition suggested something decidedly more severe than a frontal sinus abscess. Examination found the septum deflected to the left, which made it impossible to catheterize the frontal sinus. A mucopurulent secretion was found oozing from the middle meatus. Under cocain the anterior half of the middle turbinal was easily removed, and by cauterizing the discharge was found to come from the frontal sinus. The patient seemed decidedly better for a short time after the removal of the middle turbinal, but very shortly the frontal pain returned.

It was decided that there was probably a brain abscess. Several neurologists examined the case and agreed with the diagnosis, but were unable to locate the abscess. There was no middle ear trouble, and since the frontal sinus was infected, it seemed probable that the frontal sinus had some relation to this infection. After opening the frontal sinus the condition found did not indicate a process which was likely to invade the brain cavity. There was no perforation of the posterior wall of the sinus. However, it was thought best to remove the posterior wall in order to make sure that there was not an extradural abscess in this region. None was found. Still believing that there was a brain abscess and probably located in the frontal lobe, an opening was made higher up and the bone trephined. No abscess was found. After about a week symptoms of right sided paresis appeared, which pointed toward a probable temporosphenoidal abscess. The bone was trephined and a large abscess was found in this region. The colon bacillus seemed to be the active organism.

An interesting feature of the case was that the patient showed marked aphasia for names of objects; while recognizing the object itself, he could not name it. The patient was left handed, and the abscess was located on the left side, but the location of the speech center in his case was on the affected side—the left side—because he had been taught to do one thing with his right hand, and that was to write.

Dr. Otto T. Freer did not think that Dr. Shambaugh was clear in the pathology in the case he reported, and he especially failed to show a connection between the frontal sinus opened and the abscess found. While there are cases where, by way of lymphatic infection through bone, brain abscess has been produced, these cases are in the nature of curiosities, and brain abscess due to frontal sinus suppuration practically always implies ulceration of the mucous membrane of the posterior wall of the sinus, caries of the bone and infection of the dura.

Dr. Freer did not regard the signs which led Dr. Shambaugh to a diagnosis of frontal sinus suppuration as enough to warrant such a diagnosis. Pus in the middle meatus may mean suppuration of the antrum of Highmore or anterior ethmoidal cells, instead of suppuration of the frontal sinus, for the pus from all these cavities appears in the middle meatus. Pain over the frontal sinus is a very uncertain sign of frontal sinusitis, for it is often due to supraorbital neuralgia, and even oftener, in Dr. Freer's experience, is reflected pain due to suppuration of the maxillary antrum, with a healthy frontal sinus underneath the region of the pain. Dr. Freer has often seen rhinologists misled by brow pain into a diagnosis of frontal sinus suppuration, when the disease was a suppuration of the maxillary antrum. Dr. Charles E. Parker, of the Golden Square Hospital, London, and Dr. Brown Kelly, of Glasgow, informed Dr. Freer that pain over the frontal sinus, in their experience, was very often caused by maxillary sinusitis with a healthy frontal sinus.

Dr. J. R. Fletcher had seen a large abscess of the frontal lobe emanating from frontal sinus empyema on the right side, but the relatives would not allow an autopsy.

Dr. Clark W. Hawley reported a case that he operated on last summer. An extensive mastoid dissection was done and quite a portion of the middle fossa and the lateral sinus was uncovered. The convalescence was very slow at first, but he began to show all the signs of brain abscess, temperature down to 94°, pulse to 54, weakness and other symptoms. Several consultations were had. It was decided that the indications were not sufficiently well marked to justify opening the skull, and the result proved the wisdom of it. From this relapse he seemed to be recovering, when he suddenly died one night. A postmortem was done, and the most careful dissection of the brain showed no abnormal change; no abscess or meningitis could be demonstrated. This shows that there are two sides to the subject, and that we cannot always depend on lowered pulse rate and temperature as indicative of brain abscess.

Double Fistula (Right and Left), the Right Being Probably in the Horizontal Semicircular Canal, the Left in the Oval Window.

Dr. J. R. Fletcher demonstrated two cases, as follows: First, that of a man, about forty-five years of age, with a double fistula (right and left), the right being probably in the horizontal semicircular canal, the left in the oval window. The drumheads were both adherent to the labyrinth wall. The fistula left was plainly visible. The man had complained of vertigo when blowing his nose. This symptom caused him to consult Dr. Fletcher. Knowledge of the fistula symptom directs one's attention immediately to the ear, although this man did not complain of his ear at all. Examination revealed cholesteatoma in the external auditory canals, both right and left. The interesting feature about the right fistula was that in doing the Valsalva he developed pronounced vertigo, with true vestibular nystagmus toward the diseased side. Inspection of the drumhead at the time of the blowing showed a distinct track which the air followed. This was located along the inferior margin of the drum. No change took place in the left drumhead. Both sides responded to the fistula test, each causing nystagmus to the right. There is as yet no satisfactory explanation why the fistula test in one case makes a nystagmus in a certain direction, and in another case in the opposite direction; in other words, we cannot explain the lack of constancy in the direction of the flow of the endolymph in the canal under seemingly like circumstances. Before the removal of the cholesteatomata there was no response to the fistula test. It seems to Dr. Fletcher that this case shows clearly the value of the fistula symptom.

The second case was one of complete

Destruction of the Function of the Right Labyrinth Following a Radical Mastoid Operation.

There is a fistula present in the lateral arm of the right horizontal semicircular canal, which can be easily seen. The excretion from this fistula is serum. This may be a case of serous labyrinthitis following the radical mastoid. As Dr. Fletcher did not do the operation, and the history previous to the operation is not clear, positive statement cannot be made. The case is interesting because the patient responds very faintly to the turning and the caloric tests of the good ear, much more slightly to the turning test, and not at all to the caloric test of the bad ear. This case illustrates the readjustment which takes place after the functional destruction of one labyrinth, namely, the assumption of the function of both labyrinths by the single good one. The use of the Bárány noise apparatus for the exclusion of the good ear was also demonstrated in this case. During its use the patient was totally unable to hear very loud spoken language.

Paper: Clinical Vestibular Nystagmus.

BY JOHN R. FLETCHER, M. D.

DR. FLETCHER said it is quite important, in familiarizing ourselves with the use of vestibular nystagmus in otology, to remember that there are many cases which do not come under the rules, and that all cases of this type of nystagmus do not originate in the ears—that is, they are extracranial, as, for instance, the nystagmus with vertigo from auto- and other intoxication, uterine vertigo, etc. No direct ear symptoms and none pointing directly to the cranial contents will speedily put us right on these cases. Other cases, manifestly of the gravest, are at present quite beyond diagnostic certainty and are helped but little by the symptom under consideration. However, in diagnosing and differentiating serous and suppurative, circumscribed and diffuse, manifest and latent labyrinthitis; hyperemia and anemia of and hemorrhage within the labyrinth; and retrolabyrinthine disease from one another and from nonotologic diseases, its place is well fixed and its value great. The patients we see are referred or come of their own accord; we are asked to see the first because of the nystagmus, and are consulted by the second because they have vertigo. They know nothing of the nystagmus, but for various reasons believe their vertigo comes from the ear or nose; as, for instance, the first patient demonstrated by him that evening, who came complaining of brief attacks of severe vertigo, with sometimes nausea, whenever he blew his nose. Careful examination determined beyond doubt that he had a fistula in both ears, which is uncommon, and, further, that but one of them is affected by the nose blowing. We learn the latter from the fact that performing the fistula test on the left side produces nystagmus in the same direction as by blowing the nose, but its velocity and excursion are not so great. If we make the same test on the right, the result is identical with the nose blowing. This and the unique fact that the route of the air current from the eustachian tube can be followed with the eye make it certain. In addition, a pinhole perforation of the closely adherent left drumhead, directly over the oval window, designates the site of the fistula. Moreover, this perforation would allow air coming from the tube to escape. Another point to make is that gentle pressure over the site of the perforation with a probe wound with cotton causes nystagmus, much the same as the fistula test. The latter is made by carefully blowing air into the external auditory canal through a hollow olive-shaped hard rubber point from a small rubber bag and tube. The fact that his hearing is good beyond expectation signifies that the disease is circumscribed. Air can be prevented from escaping through the eustachian tube when it is wide open and the drum membrane is perforated, by the patient holding his nose and blowing at the time the test is made, as suggested by Ruttin, or by closing the tube with cotton within the middle ear, when possible.

Of the three clinical tests, the fistula test, turning, and the caloric test, the latter is the most constantly useful. The speaker's second patient brings out this point quite well. Her response to the fistula test is negative, though one is visible on the right, and freely exposed to the pressure of air because of tubal closure after the radical mastoid operation. She is totally deaf in this side. Schwabach localized in the left; Rinné negative +. She responds very slightly to turning, either to right or left—so slightly that unless very closely observed one might report the response absent. Her response to the caloric test on the right is negative. All these facts signify beyond a doubt total functional inactivity—a functional death of the right labyrinth. The whole exposed lateral labyrinth wall was irrigated with ice water for a long time without result. The same water used in the left ear caused horizontal and rotatory nystagmus to the diseased side, but not so promptly nor so vigorously as in the normal person. Quite warm water caused to flow into the left ear caused a rather feeble rotatory nystagmus to the good or irrigated ear. These caloric tests to the left ear tell us that her left labyrinth is functionating. If we had no other information than the caloric test given us, we could safely diagnose a dead labyrinth on the right and a functionating one on the left, though the left response is normal only in the direction and plane of the nystagmus, not in quickness and degree. In other words, what is known as readjustment has taken place —the good labyrinth is somewhat less keenly doing the work of both. Their action and reaction on each other has been lost—there is no longer synchronysm. The normal reaction to turning a patient ten times around the vertical axis is nystagmus in the direction of turning during turning, and in the opposite direction after turning. In turning this patient to the right there is no after-nystagmus, and the usual vertigo is absent. In turning to the left the after-nystagmus is so slight as to amount almost to nothing. When we use the turning test to discover pathologic labyrinthine conditions, the rule is that if one labyrinth gives an after-nystagmus of at most half the duration of the other, the labyrinth giving the short-enduring reaction is pathologic. In this case one does not respond at all and the other barely. We may interpret this as nothing, equaling the half or less of almost nothing; that is to say, within the rule. Turning must always be used in case of dry perforations, as irrigation is not to be thought of on account of the danger of causing acute suppuration. Of course, the caloric test may be made with clean air, warm or cold, or any other cleanly method which may be devised to change the temperature of the labyrinth wall, except the use of fluids, as above. The turning tests of Dr. Fletcher's first patient have not been made, as he objects strongly to the vertigo.

Let us suppose a patient complains of vertigo coming on in attacks of from a few seconds to a few hours' duration, of a light or relatively severe grade, with the history of an old chronic suppurative otitis media. We must examine for spontaneous nystagmus by directing the patient to look to the extreme right and left without turning the head. If in this eye position we find nystagmus directed to one or both sides, we may regard the case as circumscribed labyrinthitis; but to make the diagnosis more certain the caloric reaction should be positive, with change of direction of the spontaneous nystagmus to the use of cold when to the right, and of warm water when to the left. There should be a response to the turning test, with the comparative difference mentioned above. The hearing test should be positive. The fistula test should be made, and will usually be found positive; though a fistula may be present without a positive response to the test, due either to the escape of the air or to some obstruction to pressure, such as cholesteatoma, granulations, etc. Diffuse serous labyrinthitis occurs in acute suppurative otitis, chronic suppurative otitis media, and follows the radical mastoid operation. The characteristic nystagmus is toward the sound side, rotatory and severe. Not infrequently the circumscribed changes to diffuse serous or diffuse suppurative labyrinthitis. The question of a labyrinth operation is thrust before us, and is settled by the differential diagnosis. In both cases the nystagmus will be directed to the sound side and is severe. If any sign of functional activity of the diseased side remains, namely, response to the caloric, turning, fistula and hearing tests, the case is one of serous labyrinthitis. Diffuse suppurative labyrinthitis is totally destructive and very dangerous to the cranial contents. In such case there is no response to tests, and so far as they are concerned we must diagnose diffuse suppurative labyrinthitis. Unfortunately, we have reports of operated cases in which all reactions failed which were not suppurative, and others in which the labyrinth operation was done which recovered. We must, therefore, conclude that the serous labyrinthitis sometimes causes the picture of diffuse suppurative. These cases cannot be clinically differentiated from the diffuse suppurative. There is a great deal more to be said about serous labyrinthitis, but as we are dealing more particularly with the symptom—nystagmus—from the clinical standpoint, it may be well not to go too far afield, but proceed to the next and most dangerous disease of the labyrinth, namely, diffuse suppurative labyrinthitis.

No picture is quite the same as that of the serous. Severe vertigo, nystagmus to the sound side, nausea, vomiting, and disturbance of equilibrium. This is the manifest type. Latent diffuse suppurative labyrinthitis progresses more slowly. The history will show complaint of attacks of vertigo at intervals of greater or less time through the course of years. There will be no response to any of the function tests of the labyrinth —hearing test negative, caloric test negative, turning test negative, and no fistula symptoms. The only possible symptoms are, first, very small spontaneous rotatory nystagmus, both right and left, when the glance is in these respective extreme directions. Second, the marked difference in the duration of the after-nystagmus following turning to the right and left, head erect. Ruttin gives the difference as from one to three to six. Let us suppose the right labyrinth to be the seat of the trouble—so long as true compensation, as defined by Ruttin (Klinik der Serösen und eitrigen Labyrinth-Entzündungen), has not taken place, the result of turning ten times to the right, head erect, will be an after-nystagmus to the left of from fifteen to thirty seconds' duration, while turning to the left, used in like conditions, created an after-nystagmus of about five seconds' duration. When complete compensation has taken place, that is, when not the least particle of function remains in the diseased labyrinth, the sound side assumes the function of both sides, but somewhat less acutely, as follows: The after-nystagmus following ten turnings, either to the right or left, head erect, in the opposite direction to turning for ten to fifteen seconds. In other words, the after-nystagmus again becomes equal to both sides, differing from the normal in that its duration is reduced to one-half or one-third or less. The sound labyrinth does all of the work, but is only able to produce half the result in point of duration. Several authors have reported necrosis with sequestration of a part or the whole of the labyrinth. These cases, of course, are latent suppurative labyrinthitis, and sequestration may be considered the last symptom, unless we add facial paralysis, which now takes place. Ruttin mentions two cases in the book above named in which the fistula symptom was present after sequestration, showing that some filaments of the nervous end organ yet remained. The early picture seen in traumatic destruction or hemorrhage into one labyrinth is quite the same as in diffuse suppurative and extreme serous labyrinthitis, but here we are not dealing with a case of old suppurative otitis media, while circumscribed serous and suppurative labyrinthitis of necessity follows otitis media. On the other hand, it must not be forgotten that an otitis media may follow the trauma. The trauma caused by the radical mastoid operation not infrequently converts a circumscribed into a diffuse serous labyrinthitis. The nystagmus characterizing (though we rarely see it) the circumscribed labyrinthitis should not change after the radical operation. If it does, it is spontaneous rotatory and directed to the sound side. The great question, then, to be decided is between the serous and suppurative diffuse labyrinthitis. Clinically, today we must consider all cases showing no reaction to the fistula symptom, hearing, caloric and turning tests, with strong nystagmus to the sound side following chronic suppurative otitis media, diffuse suppurative labyrinthitis. The accompanying symptoms of nausea, vomiting and disturbances of equilibrium are, of course, usually present.

While the above relating to nystagmus is the rule, in a considerable number of cases of suppurative labyrinth destruction there is a horizontal nystagmus to the diseased side. Further, the nystagmus to the sound side may be compound; that is, both horizontal and rotatory. When there is a nystagmus to both sides by glancing in the extreme lateral direction, one may be confused who thinks of the rule that nystagmus is increased when the patient glances toward the quick component and decreases or stops completely when the glance is in the opposite direction. An important fact is that the nystagmus emanating from the labyrinth is strongest at the beginning, gradually growing weaker and weaker until it ceases completely.

It is difficult to write of clinical nystagmus and keep strictly to the text when it comes to the consideration of serous and suppurative labyrinthitis, because the nystagmus is the same in both cases during the time when the differential diagnosis must be made, if possible, though the attempt has been made to direct attention to the nystagmus throughout the paper. The real danger to the life of the patient is through intracranial, that is, extralabyrinthine, complications, of which there are two in particular, from labyrinthitis, abscess and meningitis. Fortunately the nystagmus serves us here, and that, too, in the midst of the symptom storm.

In cerebellar abscess the nystagmus may be directed to the sound as well as to the diseased side. Characteristic for this nystagmus is it that as the disease progresses the nystagmus becomes more intense, ultimately reaching such a grade as is never seen in labyrinth disease.* In labyrinth disease the nystagmus to the sound side predominates and, last, after that which may have been directed to the diseased side ceases. If this nystagmus suddenly changes to the sound side and is strong, with tendency to increase rather than diminish, cerebellar abscess may be diagnosed confidently. Other cases may be doubtful, but this may be said with certainty, that if the complete labyrinth operation has been done and nystagmus persist to the diseased side, there is an intracranial complication. (Physiologie und Pathologie (Functions-Prefüng) des Boyengang-Apparates beim Menschen.) Bárány says: "Is the right vestibular apparatus unirritable, with a strong rotatory nystagmus to the right, so is the same caused by an intracranial disease." A dead labyrinth cannot originate a nystagmus toward its own side; therefore, the irritation must come from somewhere along the course of the vestibular nerve or its center.

---

*H. Neumann: Zur Differential Diagnose von Kleinhirnabszess und Labyrintheiterung.

The differential diagnosis between cerebellar abscess and meningitis of the posterior fossa cannot be made by means of the nystagmus. It is, in fact, usually difficult, though the other symptoms are the real guide and frequently do not fail us.

To those who wish to follow the subject more deeply, Neumann's "Der Otitische Kleinhirnabszess" will be of great value.

DISCUSSION.

Dr. William L. Ballenger said the average otologist was very much confused by the theoretical problems presented in a study of this question. As a matter of fact, there is one class of cases at least that can be diagnosed without knowing anything about nystagmus, as, for example, latent diffuse suppurative labyrinthitis, with total deafness and a history of a vertiginous attack at one time in the past—it may be weeks, months or years previously—and nystagmus might have been observed at that time. Since that time there has been total deafness and a discharging ear. Those cases are referred to as diffuse suppurative latent labyrinthitis. One might be able to make a diagnosis of a case of this sort, even if he has not made a study of the deeper problems presented by Dr. Fletcher. It is easy for an otologist to know that he has a case of total destruction of the labyrinth. If we have, then, a case with a chronic discharge and the patient more or less deaf, the noise apparatus, the stream of water, or the tuning fork, as suggested by Dr. Pierce, inserted into the meatus, we can determine whether a patient has total destruction or not, and then, with a previous history of a vertiginous attack, we can arrive at a fair estimate that we have a case of diffuse suppurative latent labyrinthitis. So there is one type of case that may be fairly well diagnosed without the classical tests. Dr. Ballenger referred to a case under his observation at the present time of that type, which had been operated on ten or twelve years ago—three times within a period of a year or two—for mastoid disease. This man did not know he was totally deaf in the operated ear, and the speaker did not know it until he had been around the office for two years. He had been treating him for frontal sinus disease, and had done an operation on him for it. One day he complained of dizziness, which aroused Dr. Ballenger's suspicions. He used the noise apparatus, and found him totally deaf in the operated ear.

So the problem is not so complicated as at first thought. It is true that these more theoretical aspects of the subject must be mastered before we have complete mastery of the subject, but it has been his desire to approach the subject from the simpler standpoint. He has seen nine labyrinth cases during the past year—most of them of the latent type, with a history of vertiginous attacks eight or ten years previously. Ruttin says that when the labyrinth has been long destroyed we may get complete compensation; that is, the nystagmus after the turning test is equal in either direction, though of shorter duration than in normal cases.

Dr. Norval H. Pierce said that Dr. Fletcher had presented the known facts that can be used in clinical work very well. He did not think he had anything to add. He carries out the examinations about as Dr. Fletcher does.

Dr. Robert Sonnenschein asked whether a Wassermann had been made in the case of the woman who had lost the reaction to turning, but still had the caloric. At a meeting of the Vienna Otological Society, held about two years ago, Neumann stated that the fact of loss of turning reaction and retention of caloric reaction almost always suggests lues.

Dr. Fletcher said the woman had had an abdominal trouble for which she was operated. A Wassermann was made and was negative.

Dr. George E. Shambaugh stated that there is usually no difficulty in recognizing an involvement of the labyrinth in connection with suppurative middle ear disease. If the labyrinth involvement develops suddenly, there are symptoms of disturbed equilibrium, including nystagmus, which are always present. If the labyrinth involvement is one of long standing, especially where there has been a destructive process, the symptoms of disturbed equilibrium may be largely absent, but in these cases the labyrinth involvement can be diagnosed by the fact that the function of the labyrinth on that side is destroyed. In these cases of labyrinth involvement there may be some confusion in deciding on the proper treatment. Dr. Shambaugh thinks that the most important thing clinically is to determine when an operation on the labyrinth is indicated and when it is not. In all cases of acute labyrinthitis, where the disease stops short of a complete destruction of the function of the labyrinth, all are at present agreed that no operation upon the labyrinth is called for. These cases are usually classified as cases of serous labyrinthitis, and unless they develop into diffuse suppuration of the labyrinth, the development of an intracranial complication is extremely unlikely. It is only in cases of diffuse suppuration of the labyrinth, where there is already a complete destruction of the labyrinth function, that the question of a labyrinth operation is at all considered. In view of the large number of cases which recover spontaneously after a diffuse suppuration of the labyrinth, the question naturally arises whether we are able to make a distinction clinically between the cases of diffuse suppuration of the labyrinth which call for a labyrinth operation and those in which a labyrinth operation may not be performed.

In examining cases where there has been a long standing purulent otitis media, one frequently discovers a totally dead labyrinth. It is not necessary to subject all of these cases to a labyrinth operation. It seems probable that only those cases of chronic purulent otitis media, where the indications exist for a radical mastoid operation, that is, where there is an invasion of the temporal bone, with or without cholesteatoma formation, that the labyrinth operation should be performed. The question may not be so simple in cases where one observes in purulent otitis media the development of an acute labyrinth suppuration. There are some who believe that in all of these cases the labyrinth operation should be immediately performed. The speaker has not been able to subscribe completely to this opinion. He referred to a case that he had observed over a year ago, who had what appeared clinically to be a mild acute otitis media with no severe pain and very slight rise in temperature. The patient recovered from a sudden destructive labyrinth involvement without an operation, either upon the mastoid or the labyrinth. Testing the ear one year later showed that there had been no return of function. In a similar way he had recently observed in a case of chronic purulent otitis media the sudden incomplete destruction of the vestibular part of the labyrinth. This case was not operated. The type of the middle ear suppuration clearly indicated a process which had not invaded the temporal bone. There was no evidence of caries or cholesteatoma. There was a large, central perforation and a history of recurring acute exacerbation of middle ear inflammation running over a number of years. The patient made a good recovery, but there was no return of function to the semicircular canals. The speaker believes that in many cases of acute labyrinth destruction from suppuration one may defer an operation upon the labyrinth, awaiting the development of the first symptoms indicating an intracranial extension. Of course, in cases where the clinical conditions already indicate the necessity for a mastoid operation, whether these symptoms occur in a case of chronic or in a case of acute otitis media, the mastoid operation should be performed, and at the same time the operation upon the labyrinth should be done.

Dr. Ballenger did not think it was right to let Dr. Shambaugh's statement stand. He operated on a mastoid case several years ago and did a radical mastoid. The man had total deafness at that time—had diffuse suppurative labyrinthitis. The patient lived for five years in comparatively good health, but five years after the radical mastoid he died of meningitis. It is not safe to allow such cases to go without a radical labyrinth operation. Sooner or later meningitis or brain abscess is liable to occur. The labyrinth operation can do no harm, provided the operator knows the technic. We would not allow a chronic mastoiditis to go on indefinitely without operation, and there is infinitely more reason why we should not allow a labyrinth total destruction to go on indefinitely without operation, as intracranial complications are always liable to occur at any time. If operated, these cases are safe in that respect.

Dr. Shambaugh stated that in the case reported by Dr. Ballenger, where there was a long standing destruction of the labyrinth, no labyrinth operation was called for unless the indications existed for a radical mastoid.

Dr. Joseph C. Beck said that the conservatism expressed by Dr. Shambaugh was certainly welcome from a man who knew so much about the question as Dr. Shambaugh. He had seen such a case as the one referred to by Dr. Shambaugh as cured that developed a very severe labyrinthitis with irritation of the dura, or meningeal irritation. He would certainly not operate on the labyrinth, but on the mastoid process.

Dr. Beck asked Dr. Fletcher whether, in the case of the man exhibited with two fistulæ, since this is an indication for operation, he would do bilateral radical operation in this case.

Dr. Shambaugh stated that in cases of acute otitis media where acute labyrinth symptoms developed, even when these symptoms indicated clearly a complete destruction of labyrinth function, he was not prepared to operate either upon the mastoid or upon the labyrinth unless indications were present for a mastoid operation, or unless the patient developed symptoms indicating an intracranial extension.

Dr. Norval H. Pierce said that it is his custom, in cases of acute or chronic otitis media, in which acute labyrinthine symptoms arise, to never operate on the mastoid for a simple mastoid or for a radical mastoid while those active symptoms were present, unless there are increasing meningeal symptoms. For proof of this he depends largely on lumbar puncture. He invariably allows these patients to remain in bed until all active symptoms have subsided, such as dizziness, vertigo, etc. In quite a series of cases he has not been disappointed in this method. Before adopting it, however, he was largely disappointed in the outcome of operations undertaken at the time of active labyrinthine irritation. He has seen fatal cases result from opening the labyrinth in the face of the active manifestations in the labyrinth, and he believes that we do not insure safety to the patient by simply opening the labyrinth. He believes that a given inflammation of sufficient virulence and clarity will proceed to the meningeal serous spaces, irrespective of whether we open the labyrinth or not. He therefore depends largely in these cases, while the labyrinth symptoms are in progress, on the chemical and cytologic examinations of the cerebrospinal fluid. In the absence of changes in the cerebrospinal fluid, the patient remains in bed at rest. They practically invariably recover from the active inflammation in the labyrinth, and then he proceeds with the radical operation, but very rarely feels called on to open the labyrinth—very rarely. He thinks the terms serous labyrinthitis and suppurative labyrinthitis are misleading in a clinical sense. They have to do largely with the character of the inflammation, and you cannot always tell at the clinic whether it is a pure serous change or whether it is a suppurative change.

The point he wished to make is that he supports the position that we should not operate in the presence of a stormy, acute labyrinthitis, whether it is serous or not, whether there is a fistula or not, unless there are indications of advancing meningeal involvement. If there are such symptoms, he would open the labyrinth and drain the subdural spaces. In a chronic otitis media with a quiescent, destroyed labyrinth and a demonstrable fistula or sequestrum, he would open the labyrinth.

Dr. J. Gordon Wilson said that he was sure the society appreciated the review given by Dr. Fletcher of the clinical applications of nystagmus. The rules given by Dr. Fletcher are of very great use. But one must recognize their limitations and desire more definite information. This, he fears, we are not likely to obtain in the first place from man. Here the conscious element is ever coming into play, modifying and sometimes making negative our findings. It has always appeared to him that we must look to the physiologist for the fundamental rules governing nystagmus.

The side of nystagmus that has interested him particularly has been the experimental. Here one sees that the fundamental facts of labyrinthine nystagmus are relatively simple. In discussing nystagmus one difficulty constantly met with is that we fail to recognize that we are referring to two different things, a slow deviation which is labyrinthine, and the quick return which is extralabyrinthine. All our tests should have this in view. Thus he would rather that Dr. Fletcher spoke of the patient with labyrinthine disease falling in the direction of the slow movement of nystagmus. In man the falling in cerebellar lesions does not follow any such definite rule.

The careful observation of nystagmus in differential diagnosis is extremely important; for instance, in the differential diagnosis between cerebellar and labyrinthine disease. Here again the fundamental rules which will guide us must come from animal experimentation. For in man so many other influences may arise to complicate results. Thus in cerebellar abscesses, for instance, we have absorption of toxins, pressure on the vestibular nerve, etc. Let us aim to get the fundamental rules; then we can learn to eliminate the unessentials.

Dr. Fletcher, in closing, referred to Dr. Ballenger's remark that "we can so easily, without these tests, diagnose a latent suppurative labyrinthitis," and later said that "he was very much astonished after a long time that the patient had vertigo." That means he never had acute labyrinthitis. He had serous labyrinthitis, because just exactly those cases recover, and the patients can again have symptoms of circumscribed labyrinthitis, such as attacks of vertigo.

Dr. Lewy asked about the action on acute suppurative otitis media in which there was a fistula present. The speaker was sure, from his own personal experience, that he could not answer this question, because had never seen a case of acute suppurative otitis media where there was a fistula present. He had seen labyrinth symptoms and vertigo, but had never been able to demonstrate a fistula in an acute case.

Dr. Shambaugh asked himself the question, Shall we operate when there is complete suppression of function, and determined that he would not. Then he referred to the case of the essayist as being a case of suppurative labyrinthitis. He would first speak about the operation.

Abroad, where they are trying to accomplish something, they maintain that we should operate, because it reduces the death rate. They have their statistics of many years' standing in which they did not operate on labyrinth cases, or very rarely. They did not recognize them—they did not know of symptoms that we now speak of as nystagmus and its accompanying symptoms. Dr. Fletcher's opinion is largely their opinion. He is inclined to follow them. What he saw when over there impressed him very much, and what he has seen since returning from there seems to agree with their statistics. The death rate is distinctly lower by operating during the stormy process; in other words, operate on a case that has been diagnosed with certainty as diffuse suppurative labyrinthitis. Bárány and all say, do not operate when it is serous. Sometimes they get brain symptoms. That is, the symptoms lead them to think that there may be a brain abscess. They operate on the labyrinth, and remove it as much as possible, and await results.

With the experience in Europe with a number of cases of serous labyrinthitis following operation, and with the fact that there is no pus coming from the fistula just now, he is inclined to think that the case is one of serous labyrinthitis rather than suppurative. He has not opened the labyrinth. In other words, while he follows the Vienna school, he also follows his own judgment somewhat. In this case he has seen no indications for an operation. He would not operate on a labyrinth because it was dead. That is a certainty.

In the discussion of what cases we should operate upon, the Vienna men say that we should operate on cases of diffuse suppurative labyrinthitis, because of the great frequency of the occurrence of meningitis and brain abscess in the unoperated cases, and also because they consider the disease more dangerous than the operation. They, on the contrary, consider the operation more dangerous than letting the disease alone, providing you do not do the complete operation. Merely opening into the labyrinth is not the labyrinth operation. They consider, then, the trauma which has been done to the labyrinth infinitely more dangerous than doing nothing, unless you open all three semicircular canals, the vestibule and the cochlea. (The Neumann operation.)

Dr. Beck asked about the other case, the one that he has not operated on, or suggested it. The Vienna school does not approve of that. Bárány especially says to beware of an operation on one ear when the other one is diseased; in other words, he says, you take a chance. This man's hearing is very good. If on either side he should develop very strong symptoms, he certainly would operate—if those symptoms were not serous. If he develops a storm and has any hearing left, or if he responds to turning or to the caloric test in the ear which produces the storm, he would still not operate. But on the first sign of developing intracranial symptoms he would operate.

Dr. Pierce asked if he would operate, in case of intracranial symptoms, whether there was function or not, to which Dr. Fletcher replied: Yes, whether there is function or not.

Replying to Dr. Wilson's remarks on the nomenclature—Bárány objects to that sort of thing. This has been discussed a good deal abroad, and they think they had better retain the present nomenclature, although the speaker quite agreed with Dr. Wilson that it would be very much better to speak of vestibular component.

ST. LOUIS MEDICAL SOCIETY.

OTO-LARYNGOLOGICAL SECTION.

Meeting of March 26, 1913.

Otitic Brain Abscess.

Dr. W. E. Sauer reported a case of an otitic brain abscess in a girl aged nineteen years, with no clinical evidence of mastoid involvement. Otitis media, discharge following paracentesis for one month, ten days after cessation sudden rise of temperature to 101,° pain over left eye, and vomiting, which continued ten days. Examination of ear at that time revealed perforation in the upper posterior quadrant, surrounded by small granulations. No sagging of the upper canal wall, no swelling or pain over mastoid. Kernig's sign present.

Operation.—Mastoid found full of pus; dura over tegmen antri exposed, also lateral sinus; no evidence of intracranial complication found. Improvement for a week. On eighth day following mastoid operation, pulse fell to 60, optic discs slightly congested; ten days after mastoid operation incision of temporal lobe for distance of 2 cm. Abscess cavity found and an ounce of pus evacuated. Patient recovered.

Intraocular Tension in Glaucoma.

Dr. H. Edward Miller reported a case of intraocular tension in glaucoma lowered by injection of Meckel's ganglion. The patient, a woman of fifty-five years, stated that ten years previous she had noticed that her right eye was almost blind and that the left was growing bad. At present time she is totally blind in right eye. She has a severe pansinusitis, accompanied with headaches and increased blurring of vision, in left eye. Following the application of cocain over the sphenopalatine foramina, Schiötz's tonometer demonstrated a drop in tension from 60 mm. to 55 mm. on the right, and from 51 to 37 mm. on the left, within forty-five minutes of its application. Myotics and nasal treatment for a few days brought the tension down to 32 mm. on the right and 26 mm. on the left. Four months later patient returned with sight much worse. Tension higher than at any time during observation —70 mm. in each eye. Mucous membrane of the nose swollen, but no sinusitis. A course of five injections into the sphenopalatine ganglion of a 5 per cent alcoholic solution of phenol was now instituted, with following results: There was an average immediate rise of 10 mm. and a subsequent average drop of 17½ mm. within twenty-four hours. The minimum drop was 7 mm. The maximum, 28 mm.

DISCUSSION.

Dr. W. H. Luedde, who observed the case with Dr. Miller, was of the opinion that the case conclusively showed the existence of a relation between nasal disease and intraocular tension. This was noted some twenty years previously by Berger, who showed, among other things, that not only painful attacks of glaucoma could be produced by diseases in the nasal fossæ, but that the application of cocain would give relief, the explanation of which, according to Berger, rests upon the possibility of reflexes being transmitted through the various communicating branches of the fifth nerve. That intraocular tension could be raised by irritation of the superior cervical sympathetic ganglion, the fibers of which accompany the fifth nerve, was demonstrated by Lodato. A commissural connection also exists between the two sides. The immediate rise of intraocular pressure caused by the alcohol injection was probably due to the irritation of the superior cervical sympathetic ganglion. The more permanent and complete nerve blocking, however, should account for the drop in intraocular tension. The commissural connection accounts for the bilateral rise and fall in intraocular tension when only one ganglion was injected. Dr. Luedde reminded of the fact that the short ciliary nerves which are distributed to the choroid and ciliary body, enter the globe posteriorly and come from the ciliary ganglion which receives a root from Meckel's ganglion. Pain impulses passing along these nerves may be blocked at Meckel's ganglion. Pain transmitted over the long ciliary nerves, as from acute iritis, could not be relieved by injection of the ganglion, as they enter directly into the first division of the fifth nerve.

Dr. Sluder reported that the exceedingly great pain that at times followed injection of alcohol into the ganglion, sometimes lasting as long as two weeks, had led him to use a 5 per cent solution of phenol in alcohol. He also reported the findings of Dr. Otto May, which appeared in the *British Medical Journal* of August 31, 1912, as follows: That alcohol injected into the Gasserian ganglion, as also into the main trunks of the fifth nerve in cats, under direct vision, was followed by almost no destruction of multipolar cells. Alcohol placed in the vicinity of the nerve trunk was destructive for the nerve fiber, but not complete unless the alcohol be placed directly within the nerve proper. Assuming that the sphenopalatine ganglion acts similarly to the Gasserian ganglion under similar circumstances, the experimental evidence, together with the fact that the sphenopalatine ganglion is not easily injected, may explain why the intraocular tension would drop and rise again.

Dr. Bliss stated that there is no direct anatomic way of explaining the pain which frequently is referred to the neck, shoulder, arm, forearm and even down to the hand, in the typical sphenopalatine neuralgia. He thought there was much room for speculation and a great deal more for work, before any definite conclusion on this subject could be arrived at.

New Tonsillectome.

Dr. J. Ellis Jennings presented a new tonsillectome designed to remove the tonsil in its capsule by the Sluder method. It consisted of the French tonsillectome mounted on a pistol-grip, the cutting blade being operated on the "pull" by means of a trigger.

ST. LOUIS MEDICAL SOCIETY.

OTO-LARYNGOLOGICAL SECTION.

Meeting of April 30, 1913.

Nerve Trunk Anesthesia and Carbolization in Nasal Surgery.

Dr. Greenfield Sluder presented a paper on nerve trunk anesthesia and carbolization in nasal surgery, in which the writer gave his experiences, extending over a period of three years, with cocain applied over the nerve trunks. His procedure was to place an applicator bearing one drop saturated solution cocain muriate (90 per cent) directly under the posterior tip of the middle turbinate, allowing it to remain fifteen minutes. A second applicator was placed over the internal nasal nerve. For the latter the writer used a smaller applicator which carried only about half a drop of a saturated solution of cocain muriate. An applicator carried blindly into the uppermost anterior aspect of the nose will usually come to rest over the slit through which the internal nasal nerve enters the nose. This nerve, being more superficial, requires less cocain. The anesthesia thus produced, in the writer's experience, gives more than a half hour's time for the performance of the most comprehensive bone surgery of the lateral nasal wall. In addition to the above form of anesthesia the writer resorts to nerve "blocking," using 5 per cent phenol in 95 per cent alcohol, injected by means of a straight needle, for both anterior nasal nerve and sphenopalatine ganglion. For the former the attempt is made to locate the slit from which the nerve emerges, and for the latter it is introduced under the posterior tip of the middle turbinate and carried into the sphenomaxillary fossa, as previously described by the writer. One-half centimeter of the solution is injected. A well-marked analgesia results which prevents to a marked degree the shock of the larger operations at the time of their performance, and the subsequent reaction, especially when the nose is packed.

Plaster Casts of the Sphenoid, Maxillary and Frontal Sinuses.

Dr. Hanau Loeb presented some plaster casts of the sphenoid, maxillary and frontal sinuses, prepared to estimate the cubical capacity and superficial area of these sinuses. The work followed a study of the anatomy of the accessory nasal sinuses from the standpoint of their greatest diameters.

The cubical capacity was found to be as follows: Sphenoid, from 0.6 to 11.8 cc., average 4.93; maxillary, from 4.5 to 24.8 cc., average 12.94; frontal, from 0.9 to 8.2 cc., average 3.58.

Superficial Area.—Sphenoid, from 2.4 to 28.2 sq. cm., average 15.92; maxillary, from 12.1 to 52.3 sq. cm., average 31.68; frontal, from 5.5 to 32.3 sq. cm., average 15.34.

ST. LOUIS MEDICAL SOCIETY,
OTO-LARYNGOLOGICAL SECTION.

Meeting of May 28, 1913.

The Endonasal Route of Attack in Hypophyseal Tumor Cases.

Dr. W. E. Sauer presented a paper under the above title, in which he reviewed the literature to date, and more especially that of Hirsch of Vienna, according to whose method he operated an acromegalic aged thirty-two years. The patient had been under observation for a period of one year, during which time very little differenec in any condition, mental or physical, was noted, with the exception of a marked decrease in the vision of the left eye. Vision right eye was 15/12; left eye, 15/200. Several months after the sella turcica decompression operation, which was done under local anesthesia, the patient's vision improved to the normal in right, and in the left to 17/40. The discussion brought out that the danger of subsequent infection and meningitis was more apparent than real, and that tearing of the mucous membrane need not be considered so seriously, especially in view of the fact that there are two permanent sphenoidal ostia through which infection could take place.

The program further consisted of a general discussion of:

(a) Form of initial incision and mode of anesthesia in submucous resection of the nasal septum.

(b) "Cut or cauterize" the inferior turbinate.

V-Shaped Cautery Tip.

Dr. Sluder showed a V-shaped cautery tip, with which, besides cauterizing, one could remove a wedge-shaped strip of mucous membrane from the entire length of the inferior turbinate, much as a small spoke-shave would.

ABSTRACTS FROM CURRENT LITERATURE.

I.—EAR.

The Radical Mastoid Operation Under Local Anesthesia.

KULENKAMPFF (*Beitrage zur klinischen Chirurgie,* Vol. 83, p. 546), in a very lengthy article, gives a detailed account of the method by which he has carried out thirty-two radical mastoid operations. All of these cases were completely successful from the standpoint that the patient did not suffer the slightest pain or inconvenience.

The technic is as follows: The patient is first given two preliminary injections of scopolomin morphin, in the usual doses. The skin behind and in front of the ear is wiped off with benzine, but not otherwise sterilized. The first point of injection is just above the concha; a 1 per cent solution of novocain, to which has been added a few drops of adrenalin, is freely injected in the loose tissue just above the periosteum, but not under it, in a backward direction, following the insertion of the concha. Without withdrawing the needle the same solution is injected in front of the ear. The second point is behind the ear, opposite the external canal. The injection is first made upward to meet the first injection and then downward toward the insertion of the s. c. m. muscle. If there is reason to believe that the process is extensive or that the mastoid tip is large, a second point is selected one inch behind the last named point and the injection made up and down, following along the hair line. The third principal injection point is at the posterior border of the external bony canal, the needle being inserted in the fold behind the ear and shoved in perpendicularly until it is under the skin of the external canal. For this purpose a 2 per cent solution is employed. When the skin of the canal has been thoroughly infiltrated, the Neumann injection into the junction between the cartilaginous and bony canal is made and the fluid slowly forced into the middle ear between the skin and the bony surface of the canal. If a perforation is present, a pledget of cotton soaked with 20 per cent alypin solution is placed in the middle ear. The field is now prepared for operation, and within ten minutes from the start the patient is ready. The author lays great stress on the fact that the patient should be made thoroughly comfortable and that the position of the head should be so arranged that the subsequent manipulation will not tire the patient.

The subsequent steps of the operation are carried out in the usual way, the author prefers to make his plastic before breaking down the bridge. The choice of chisel or forceps depends on the condition of the bone and is largely influenced by what the patient seems to stand the best. Acute mastoids, fresh attacks of old processes and intracranial complications are contraindications for this method. *Horn.*

The Bier Passive Hyperemia in Nonpurulent Affections of the Middle Ear.

STEIN (*Archiv. für Ohrenheilkunde,* Vol. 86, p. 92), in two articles, has made an exhaustive study of the physiologic and clinical features of the Bier passive hyperemia in nonsuppurative affections of the middle ear. He draws the following conclusions:

First, the employment of Bier's method possesses in nonpurulent affections of the middle ear scarcely the meaning of a well established therapeutic procedure. In special cases, however, it can perhaps be employed to assist the other therapeutic measures.

Second, subjective sensations proceeding from the ear, as regards their genesis, can be distinguished by the aid of the passive hyperemia to a certain extent. In cases in which the ear noises are diminished by the application of the band, the cause of the noises should be found in the effect of the pulsatory movements of the liquor cerebrospinalis. Such sounds arise especially in an increased blood pressure, whether caused by physiologic or pathologic processes, since it calls forth an increase of the pulsatory movements of the cerebrospinal fluid. Under all circumstances it is demanded, in cases in which the rubber band produces a lessening of the ear sounds, that an accurate internal examination should be made. Increased sensitiveness of the nervous system and pathologic changes in the sound conducting apparatus lead to an increase in the perception of the impulse proceeding from the liquor cerebrospinalis, and in this way to an origin of the ear noises.

These features can also in slightly elevated or even in normal blood pressure give rise to subjective sensations in the ear. *Harris.*

Otitis Media Acuta Due to the Bacterium Pneumoniæ Friedländer, Its Prognosis and Treatment.

Zange (*Archiv. für Ohrenheilkunde,* Vol. 89, p. 1) has had two fatal cases of acute otitis media due to the Friedländer bacillus. He describes these at length and makes the following summary:

From the cases occurring in the literature and our own observations, otitis media acuta due to the Friedländer bacillus, like the Friedländer pneumonia, presents a characteristic clinical picture, the peculiarities of which are based on the presence of the Friedländer bacillus, towit, in the enormous reproductive capacity and marked mucus production of the bacillus in the organism, associated with slight local reaction of the tissues. The middle ear secretion is from the beginning mucopurulent, microscopically very rich in mucus and bacillus, poor on the other hand in pus and fibrin. The Friedländer otitis shows often from the beginning an insidious character without markedly pronounced local symptoms, sometimes with and sometimes without fever. It is wont to run a protracted course. It leads further to severe complications of the mastoid, the adjoining parts and the intracranium, with often fatal outcome.

Complications in the same way can develop insidiously and without clearly marked symptoms. The Friedländer otitis demands, on this account, particular care in observation, and the prognosis must under the circumstances be regarded in no case as favorable. On this account it is also important as early as possible to make the diagnosis of Friedländer otitis, and thus by an early operation to be able to check its advance or the fatal results of severe complications.

To this end it is recommended that in every bacteriologic examination of the secretion from the ear, the staining of the capsule should be the principal thing. For this thionin is admirably adapted, which, as is already known, shows very well the capsule of the streptococcus mucosus. *Harris.*

Sudden Deafness From Scarlet Fever Otitis, and the Recognition of Otitis Interna Serosa.

MANASSE (*Archiv. für Ohrenheilkunde,* Vol. 89, p. 146) gives a valuable contribution to this important subject. Up to the present any accurate description of the microscopic changes occurring in the middle and inner ears as a result of scarlet fever otitis is lacking. Politzer in the last edition of his book reports no case microscopically examined directly after the onset of the deafness. Manasse describes two cases occurring in his practice, of scarlet fever deafness, which recovered as far as the general condition was concerned, although the hearing was not improved, and a third case which went to autopsy. Of this latter case he has made a careful microscopic study and gives the findings, together with illustrations.

The alterations which took place in the middle and inner ear he summarizes as follows:

On both sides there was a typical acute necrosing otitis media scarlatinosa. On the left side there was a total destruction of the ligamentum annulari and of the stapes; purulent inflammation of the membrana tympana secundaria with a small fistulous perforation; otitis interna purulenta diffusa with ostitis acuta of the labyrinth capsule. On the right side there was total destruction of the membrana tympana secundaria; purulent inflammation of the ligamentum annulari without perforation; otitis interna purulenta circumscripta in both windows; otitis interna serosa diffusa of the membranous labyrinth; beginning acute ostitis of the bony semicircular canal; degenerative atrophy of the cochlea.

There are a few cases in the literature of microscopic examinations of the inner ear in scarlet fever otitis. None of these, however, were made immediately after the onset of the attack. Manasse is of the opinion that the perforation through the left round window was the cause of the sudden deafness in his case. He raises the interesting question whether in the first two cases, clinically but not anatomically examined, the pathologic condition was not the same. In all three cases we have to do with grave scarlet fever diphtheria, double diphtheria, scarlet fever otitis, characterized by a necrosis of the soft parts and of the bone. All three cases show the same degree of sudden complete deafness following hearing relatively well preserved. Each of the two cases which are alive has passed over into deafmutism.

In view of these facts, he is of the opinion that the anatomic changes were similar in all three cases, although he would not claim that in all cases of scarlet fever deafness the process is the same and that there cannot be an involvement of the auditory nerve. All cases of complete deafness following scarlet fever otitis are not necessarily permanent, as is shown by a case which he reports, where the hearing following a radical mastoid operation returned, and the patient is now able to hear the conversational voice at a distance of from five to six meters.

The article concludes with the anatomic findings in the case of a patient seventeen years old, who had suffered for fifteen years from a purulent affection of the middle ear, the result of an attack of scarlet fever. The case was operated upon for mastoiditis and brain abscess, and the findings in the middle and inner ear are accurately described. Those of the inner ear are especially interesting. They include an extensive atrophic degeneration of the nerve elements, especially of the organ of Corti, the ganglia spirale, and of the fine terminal branches in the cochlea. He believes that all the changes in the middle ear and labyrinth must be ascribed to a chronic inflammation of the soft parts and of the bone, which has run its course. He admits, however, that the interval of time elapsing does not allow a positive opinion, but he is inclined to believe that in this case, and in any other similar case, we are warranted in speaking of a healed otitis internus serosa.

Harris.

The Relation of the Ostium Tympanum of the Tubæ Eustachii to the Fundus of the Cavum Tympani, as Well as to the Foramen Rotundum and the Foramen Ovali.

Zytowitsch (*Archiv für Ohrenheilkunde,* Vol. 89, p. 280) has by means of a specially constructed apparatus been able to make accurate measurements on the dry skull of the landmarks in question. Among the interesting deductions from his measurements which he makes are the following:

That the ostium tubæ eustachii in a horizontal position of the skull is only 3.7 mm. higher than the floor of the cavum tympani and almost on a level with the middle of the niche of the foramen rotundum; the foramen ovali is 3.7 mm. higher than the ostium tubæ eustachii. As compared with the antrum mastoidæ, the tubæ eustachii lies considerably deeper. By inclination of the skull fifteen degrees forward, these relations change and the tubæ eustachii becomes lower. In general, the ostium tubæ eustachii has a position extremely favorable for the drainage of fluid from the middle ear toward the nasopharynx. As a result of these measurements he draws the following conclusions:

First, (a) the tubæ eustachii functionates as a ventilation canal for the middle ear; (b) if there is secretion present in the tympanic cavity, the eustachian tube offers a means of escape for it. Second, in the presence of symptoms of acute otitis the patient must be instructed to bend the head as far as possible downward and to the shoulder on the healthy side. Forcible respiration through the nose has a distinctly beneficial effect in restoring the patentcy of the eustachian tube, thereby assisting in the removal of the secretion from the middle ear. *Harris.*

A Case of Erosion of the Sinus With Embolus and Pyemia, With a Fatal Result.

SCHLAGEL (*Archiv. für Ohrenheilkunde,* Vol. 90, p. 45). The patient was a man of 29 years, operated upon for chronic middle ear suppuration. The operation revealed the sinus lying far forward. As the result, at the outset a slight wounding of the wall took place. Two weeks after the operation a severe hemorrhage took place, and the next day, when the dressing was removed, an embolus through the vein took place. In spite of a ligation of the jugular, it was impossible to control the development of the pyemic process, and the patient died a month after the operation. The author considers some of the questions raised by his case. Erosion of the sinus is not an exceedingly rare occurrence. Twenty cases up to 1905 were reported in the literature, of which only six came to operation. An embolus was the most interesting phenomenon. He dwells on the fact that it could have taken place and not been followed by a fatal result.

He considers especially the three questions as bearing upon the fatal result: first, of the exposure of the sinus; second, the ligation of the jugular; third, the failure to clean out the bulb. He admits the unfortunate result of having bared the sinus, but insists that under the circumstances it was impossible to avoid it. There was, in his opinion, no occasion for a ligation of the jugular at an earlier time. As regards the bulbous operation, there appears to be a divided sentiment. Personally he doubts if the operation would have had any effect, however. *Harris.*

The Whispered Voice and Conversational Speech in Their Relation to One Another.

JULIUS VEIS (*Archiv. für Ohrenheilkunde,* Vol. 90, p. 200) has made an extensive comparison of the whispered voice and the conversational speech in various forms of ear trouble, and as a result of these investigations he is of the opinion, first, that the testing of the spoken voice as well as the whispered voice in every case of marked lowering of the hearing (whispered voice under one meter) is to be made from a diagnostic, therapeutic and prognostic standpoint. The testing with the whispered voice alone does not give a satisfactory picture of the acuteness of hearing. Secondly, in otosclerosis and in many cases of pure middle ear suppuration, the conversational voice (deep tones) is not heard much further than the whispered voice (high tones), while, on the other hand, nervous hard hearing and in the exudative middle ear processes, the conversational voice will be heard much better, comparatively, than the whispered voice. Thirdly, when the conversational voice is markedly better than the whispered voice, there is a decidedly better outlook as regards success in treatment. Fourth, the improvement of the hearing by air inflation many times affects only the conversational voice, while the whispered voice is only slightly or not at all.

Harris.

Plastic Operation for Congenital Malformation of the Hearing Organ.

KOSOKABE (*Archiv. für Ohrenheilkunde,* Vol. 90, p. 214) reports two cases operated upon for deformity of the auricle and external auditory canal. Kaufmann's observations are referred to in such cases: First, that the tuba eustachii is for the most part irregular. Second, the bony wall of the auditory canal is lacking. Third, the temporal bone is often deformed. Fourth, the drum membrane and the tympanum are not present. Fifth, the labyrinth windows and the stapes are for the most part normal. Sixth, men and women were equally affected.

The indications for operating in such cases he gives as follows: First, when it is possible to show the existence of any hearing; second, when stimulation of the vestibular apparatus is positive; third, when a cosmetic operation is urgently desired.

As a result of his study of the two cases, he gives the following results: First, by operation the lower tone limit is extended; second, the upper tone limit is not influenced; third, there was no sound hiatus, as in Bezold's case; fourth, bone conduction is no longer than normal, and Rinné was negative, both before and after the operation; fifth, the disturbance of the hearing was similar to that produced by obstructions to the sound conducting apparatus; sixth, in both cases there was an improvement in the hearing after the operation; seventh, the middle ear and the ossicles were lacking, but the mastoid cells were well developed; eighth, the parotid gland was found lying in the space of the auditory canal; ninth, the direction of the sound was correctly given in Case 1, but not in Case 2. *Harris.*

The Acoustic Nerve and Salvarsan.

GELLE (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* October 19, 1912). After referring to the statistics of Benario, which show that in 14,000 cases treated by salvarsan there were 126 cases of neurorecidive, and of these 126 cases, 62 had involvement of the acoustic nerve; and of Valentin, who reports 45 cases of lesions of the auditory nerve after the injection of salvarsan, he reports three cases occurring in his own practice.

The first was a case of recent syphilis which had been treated by salvarsan with satisfactory results. Three injections were given at intervals of a week. Fifty days after the first injection deafness appeared in the left ear. Another injection of salvarsan was given. Three days later deafness in the right ear resulted. The deafness in the right ear entirely disappeared after seven days, but persisted in the left ear. The Wassermann in this case remained positive.

The second case was a young man of twenty-four years of age, where the initial lesion had been contracted two months before. Sixty-three days after the first injection and forty-six days after the last injection (three injections were given in all), a right facial paralysis of peripheral origin resulted, followed by paralysis in the right cochlea and the vestibular nerve. Two additional intravenous injections of salvarsan were given after the development of the paralysis, but without effect. Here also the Wassermann reaction remained positive.

The third case was a man of twenty-seven years of age with a history of syphilis of one year's duration. Salvarsan was given for the secondary skin lesions. Forty-two days after the last injection (four injections were given), there was a bilateral deafness (cochlear and vestibular nerves). On the right side the deafness disappeared in about fifteen days. It persisted on the left side.

The author feels that in these three cases there is a distinct causative relation between the salvarsan and the deafness. The toxic action of the salvarsan would not be affected by the further use of the drug. On the other hand, a mechanism due to the toxic action of insufficient trepomenes would probably be relieved by the further use of salvarsan. As will be noted in these three cases, the use of salvarsan was continued without avail. He calls attention to the curious course of the deafness. The ears were affected after an interval of some days, then at the end of eight days hearing reappeared in the ear last affected. The size of the dose seemed to have no effect, as in one case small doses were used and in another large doses. In all the cases the Wassermann reaction remained positive, and lumbar puncture always showed an albuminous fluid with pronounced leucocytosis. Two of the cases had shown a great intolerance to mercury. As a result of his experience with these three cases, the author favors the employment of mercury and the iodids in any syphilitic involvement of the ear. For nasal and laryngeal lesions he advises 606.

Harris.

Dilatation of the Jugular Bulb, Filling the Middle Ear and Part of the External Auditory Canal.

LAMMOIS (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* October 12, 1912) refers briefly to the anomalies of the jugular bulb described by Zuckerkandl. At times the bony wall is reduced to paper thinness. In a case of Odenius it formed a sufficient prominence to hide the round window and the promontory. The bone can even be so thin as to give rise to true dehiscences.

In a number of cases the jugular bulb has been wounded, in the auditory canal, usually in the course of the paracentesis of the drum membrane, sometimes in the extraction of foreign bodies, and again in the removal of the ossicles.

Lammois has recently seen a case of marked dilatation of the bulb. The patient was a woman 50 years old, suffering from suppuration of the ear, which had existed for seven years. There was an offensive odor and polyp-like granulations which suggested the possibility of cancer. Under general anesthesia the auricle was thrown forward. Inspection then showed that the membranous auditory canal was lacking, and that in the canal there was a small violet-like growth the size of a small walnut. The bony canal was found to be enlarged, with irregular borders, giving the impression of a condensing ostitis. The mastoid cells were healthy, and neither pus nor granulation were found in the antrum. Further examination of the mass showed it to be a bluish color, the appearance of a large vein, and it was easily reducible in the middle ear. There was no pulsation under the finger. A Pravaz gave black blood. The diagnosis was made of dilatation of the jugular bulb, which filled the middle ear and formed a prominent projection in the external auditory canal.

The author believes that the venous hernia was not due here to a spontaneous dehiscence or to an erosion of the blood current, but rather to a progressive caries of the hypotympanic recess. *Harris.*

A Contribution to the Study of the Reeducation of the Hearing.

RICHEZ (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie*, November 9, 1912). The failure of all forms of treatment for otosclerosis is generally recognized. One cannot prevent the progress of the disease, but he can strive to awaken a dormant organ and reeducate it by appropriate exercises, just as one restores suppleness by means of massage and anchylosed articulation. The idea of reeducation is not new. Urbantschitsch was the first to demonstrate this by employing an accordion, but this method was not satisfactory. Zund-Burguet was the first to see the desirability of employing for this purpose the human voice, because of its richness in the low harmonics. He showed, however, that for manifest reasons it cannot be employed in the education of the deaf, and accordingly has devised an apparatus to take its place.

The apparatus is called the electro-vociphone, and is capable of reproducing the sounds which the human larynx can give forth. The agent is a number of layers of cotton, making a true artificial larynx corresponding to the three registers, the low, medium and high, and put in vibration by a current of from four to six volts. The intensity of the sound can be instantly varied according to need, and if one of the two ears is more sensitive than the other, it can be treated apart from the other.

The author has employed the apparatus with surprising results, and is thoroughly convinced of its value. He gives a report of six cases in his practice where it has been used with success. In conclusion he raises certain important questions, first, as to the permanency of the cure. His first case contracted sore throat with a temporary reduction of the hearing. With the disappearance, however, of the angina, the hearing returned and became as good as formerly. In the experience of all who have used it, the results are maintained at the end of three or four years.

In answer to the question, "What is the variety of deafness suitable for the treatment?" he replies, "Especially cases of sclerosis where the deafness has come on insidiously or as the result of confinement, or grippe, or of an infectious disease." It is indicated also in cases of adhesive otitis and in chronic middle ear catarrh the result of nasopharyngitis, although in these cases the results will be much better if treatment of the nose and throat is undertaken at the same time.

Harris.

Personal Results Obtained in Otology by the Aspiration of Bier.

ERNEST BOTELLA (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie*, November and December, 1912). The use of the Bier band in otology has been attended with serious consequences. On this account, Botella has substituted aspiration for it. For this purpose a Politizer bag or syringe can be employed. He makes use of curved glass tubes having an olive-shaped head at the end. These are of different sizes and are introduced deeply into the canal. A rubber tube connects the canula with a strong rubber bag. By these means he has obtained most satisfactory results. In a series of ten cases of ear suppuration which he reports, four were caries of the attic, in which although a cure was not obtained there was considerable improvement; two were cases of caries of the attic which were cured; two were cases of acute otitis media with mastoiditis, which were cured; one was a case of chronic otitis media, cured; and one was a case of large polypus of the tympanum, where all treatment was discontinued at the second visit.

Aspiration has the double advantage of removing the secretion from the tympanum and the adjacent cavities, and secondly, of producing an active hyperemia in the parts affected. Its curative action depends upon a phagocytosis and a reconstitution by an increase of the vitality, rather than by accumulation of antitoxin as produced in the ordinary method of Bier.

He reaches the following conclusions: First, that the direct aspiration of the auditory canal is the best means of evacuating the secretions from the middle ear; second, in order to avoid the inconvenience which can be produced by pressure on the walls of the canal, canulæ are to be recommended, carried well into the canal; third, the perforation of the drum is to be carefully watched in order that it may be enlarged if it has a tendency to close before the diseased process is cured; fourth, although aspiration can be employed without danger in all cases, the results are most favorable in simple acute otitis media with mastoiditis, and simple chronic otitis media; fifth, aspiration for the removal of granulations and small polypi is superior to the curette, because its action reaches regions where the curette cannot touch; sixth, it can be tried in cases of adhesive otitis and generally in all cases where it is thought necessary to counterbalance the effects of the external pressure upon the tympanum and the ossicular chain; seventh, the treatment should last from ten to twelve minutes at the maximum, it should be stopped if blood follows, and should be carried out gently in order to prevent nausea or pronounced vertigo. *Harris.*

Acute Otogenous Meningoencephalitis.

G. ALEXANDER (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie,* November and December, 1912) is of the opinion that meningitis is not a distinct entity, but that there exist different forms of meningitis; as, for instance, traumatic, ophthalmic, rhinologic., etc., each different distinctly from the otitic.

He divides meningitis of the ear into five groups: First, meningitis in acute suppurative otitis media without other complications; second, meningitis in chronic suppurative otitis media without complication; third, meningitis in cerebral abscess of otogenous origin; fourth, meningitis in thrombophlebitis of otogenous origin, and extradural abscess; fifth, meningitis in suppuration of the labyrinth. He further divides meningitis into early and late meningitis. He then gives the report in detail of seven cases of meningitis, all cured by operation. The first case was a child of four and one-half years of age, suffering from serous meningitis following acute suppuration of the middle ear; the second case was also a child of two years, where the meningitis followed a chronic suppuration of the middle ear. Following the operation the patient was unconscious for two weeks. The third case was a man of forty-two years of age who had suffered from acute suppuration of the left ear and osteoperiostitis of the left mastoid and serous meningitis. An anthrotomy was done and the dura exposed and incised in the middle cerebral fossa. The fourth case was a man of forty-four years of age with chronic suppuration of the middle ear, cholesteatoma, purulent (?) circumscribed meningitis. Case five was a child of nine years of age suffering from bilateral suppuration of the middle ear with cholesteatoma and acute meningitis, a cloudy but sterile spinal fluid. Case six was a chronic suppuration of the middle ear with cholesteatoma, serous meningitis, circumscribed purulent labyrinthitis; radical operation, and exposure of the dura. Case seven was a man of twenty-four years of age with chronic suppurative otitis media, cholesteatoma, suppurative labyrinthitis, acute purulent meningitis, paralysis of the external ocular motor, paralabyrinthitis, osteomyelitis of the pyramid and the base of the skull in this region; radical labyrinthine operation, a traumatic fracture at the base of the skull.

Harris.

The Residual Ear.

David G. Yates (*New York Medical Journal,* December 7, 1912) defines as residual a middle ear which has been the seat of a suppurative process which has ceased. There may be either a perforation of the drum in these cases or an intact membrane. He is of the opinion that a considerable number of cases of diving or surf-bathing, where an acute inflammation of the middle ear has been set up, belong to this class, and advances the interesting theory that many unexplained drowning accidents, in his opinion, are due to vertigo, the result of the entrance of cold water into an unprotected middle ear.

He regards as an important part of treatment preventive measures, and advises opening the mastoid when in acute cases, after free incision of the drum, postauricular tenderness persists for two or three days and there is a rise in temperature. For recent perforation, the application of a small disc of gutta percha tissue over the opening is recommended.

There may exist a necrosis in the middle ear without discharge. Once a year perhaps a few drops of pus will be evacuated. Epidermization of the edges is the chief reason for nonclosure of the apertures in the membrane. The prospect of healing of the drum is in inverse proportion to the size of the perforation. Tinnitus or marked deafness are sometimes relieved by division of bands or deposits of fibrous tissue. The writer very properly states, however, that the tendency of this adhesion is to reform. He urges the danger of the patients passing from observation before the drums are entirely healed. *Harris.*

The Radical Mastoid Operation—Some Essentials for the Proper Healing of the Wound Cavity.

Robert L. Loughran (*New York Medical Journal,* December 21, 1912). The complete removal of all areas actually diseased, or that might retard the development of healthy granulations, the establishing of satisfactory drainage, and the producing of the means of easy observation and local treatment, give the best opportunities for the rapid and permanent healing of the wound.

The author regards thorough curetting of the mouth of the eustachian tube as essential to the success of the operation.

(This is the procedure of most otologists in this country, but the Vienna school has in recent years abandoned it, leaving the tube entirely undisturbed.)

The effect of any constitutional disease upon rapid healing is dwelt upon. The author explains at length the physiology of healing of the operative cavity. Due to the greater blood supply, granulations form more rapidly in the antral wound than they do in the tympanic cavity. The development of the epidermal layer is of especial importance and depends to a large extent upon the way that the original flaps are placed. Deafness in chronic middle ear suppuration is usually due to involvement of the sound conducting apparatus. Great care is to be exercised in the preservation of the stapes on that account.

Posthealing contraction of the scar tissue over the stapes cannot be prevented, and it is this which often accounts for the frequent flagging of hearing some time after operation. Another influence as regards the preservation of the hearing is the auditory canal. While the opening needs to be enlarged, it should be done in accordance with its requirements as the conductor of sound waves. The writer properly states that the radical operation does not restore hearing to the normal, nor yet does it destroy it.

Finally, attention is called to the cosmetic results, which, in his opinion, depend upon three things: the appearance of the posterior incision and that of the concha, and the position of the auricle. The first is secured by care in making the incision and by avoiding traumatism to the edges during operation. Depression can be avoided by care in placing the incision in a location where it may have a foundation for healing; that is, on the skull surface, posterior to the margin of the wound cavity. *Harris.*

Progressive Deafness—Systemic Factors in the Etiology.

J. A. Stucky (*New York Medical Journal,* December 28, 1912) considers progressive deafness under the two divisions of nonexudative otitis media and otosclerosis. He believes that these diseases are, more than any other ear trouble, a local manifestation of the systemic condition. The subjective symptoms of otosclerosis differ very little from those due to nonexudative otitis media, except in intensity. Giddiness or vertigo occur less frequently, and seldom in aggravated form. He has collected a series of cases typical of the two diseases, which he has been able to keep under observation for from five to fifteen years, and to work out a line of treatment and method of living which has given results that justify him in the conclusion that in order to bring about favorable results it is the systemic rather than the local factor in the causative relation of these conditions with which we must deal.

The majority of cases of these diseases, in his opinion, give evidence of disturbances and faulty metabolism, indicated by the presence of endican or phosphates in the urine, or there are present indications of rheumatism. The next systemic condition most frequently observed was increased blood pressure or arterial sclerosis.

He believes that when the cure for rheumatism and arterial sclerosis is found, we shall also have solved the cure of non-exudative otitis media and otosclerosis. *Harris.*

Vertigo—A Clinical and Therapeutic Study.

Richard Lake, London (*New York Medical Journal,* January 4, 1913), divides the causes of vertigo as follows:

1. Peripheral causes. (a) Chronic progressive middle ear deafness. (b) Hemorrhage into the labyrinth, and embolism. (c) Traumatism.

2. Aural vertigo due to altered state of the blood pressure. (a) Increased blood pressure. (b) Decreased blood pressure.

3. Aural vertigo due to systemic causes. (a) Leuchemia. (b) Occasional. (c) Specific. (d) Anemic (cerebral anemia).

One of the most common causes is chronic progressive middle ear deafness. This form is of purely peripheral origin. Operation should be tried, if local and systemic measures fail. The results are immediate and certain relief. In a series of some seventy cases presenting this condition for treatment, it has been found necessary to operate on only fourteen. In each case the operation was successful as regards relief of the vertigo.

Emphasis is to be laid upon antiseptic rather than aseptic surgical measures.

In traumatic cases the vertigo rarely lasts more than six months. General measures of treatment are usually fairly successful. Two of the common but little recognized causes of this form are plugs of cerumen and the effects of violent nose-blowing. A characteristic feature of these cases is the persistence, after the disappearance of the vertigo, of an inability to walk in the dark.

The second great group of causative factors is altered blood pressure. Here also the lesion is peripheral. A peculiar feature in these cases of arterial sclerosis is the tendency of the attacks to come on in the early morning. Cases in which the blood pressure is below normal show a greatly increased vertigo on rotation. Cases due to leuchemia or pernicious anemia are not amenable to treatment. Among the most frequent of occasional causes is gouty dyspepsia. The author is of the opinion that operation is seldom needed, but when required is practically free from any danger to the life of the patient. *Harris.*

Otology From a Medical Point of View.

H. N. Spencer (*New York Medical Journal,* March 1, 1913), in a short but philosophic paper, emphasizes the importance of the otologist establishing a close connection with the laboratory. His view that wholly inadequate consideration is given to the efficiency of internal medicine in the treatment of diseases of the ear and of the nose will be generally accepted.

He urges more frequent examination of the urine and of the blood, as well as inquiries into the abnormalities of digestion. etc. *Harris.*

II.—NOSE.

Connection of the Sexual Apparatus With the Ear, Nose and Throat.

Virginius Dabney (*New York Medical Journal,* March 15, 1913) gives an excellent review of the literature on this subject and the history of eight cases occurring in his own practice illustrating the relation in hand.

His conclusion is that "many cases escape diagnosis in whom the sexual element is the contributing if not the exciting cause of many of the common nasal and aural abnormalities of function, partly for the reasons stated in his paper. partly from the lack of time needed to study intimately these cases, and partly from failure of the patients to give their full confidence." *Harris.*

Disease of the Nasal Accessory Cavities.

Seymour Oppenheimer (*New York Medical Journal,* March 15, 1913) agrees in the main with the conclusions of most rhinologists of today. His view, however, that "combined frontal and ethmoidal suppuration, with more or less polypoid degeneration of the middle turbinal, require an external radical operation for a permanent cure," would be held by most men to be extreme. This statement he qualifies, however, by adding that where there is free drainage and no signs of ill effect in the general health, the risks of the operation more than counterbalance the advantages.

He rightly states that the aim of any radical procedure is the obliteration of the affected cavity, and that if this is not accomplished, reinfection is always a possibility. *Harris.*

Treatment of Nasal Necrosis by Means of Radium.

Broeckaert (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* July 27, 1912) gives a report of four cases treated by radium with uniformly good results. He employs one cm. of the bromid of radium with a radioactivity of 500,000. It can be applied either according to the socalled destructive method which produces a necrosis of the diseased portion and subsequent cicatrix, or by the modified method which produces a slow and gradual transformation of the affected tissues.

He favors the latter method in most cases, especially in the ulcerated form of lesion, the time of exposure varying according to the extent of the lesion and the rapidity with which one wishes to produce results. The method is characterized by the remarkable uniformiiy of the results and the complete freedom from reaction.

In his hands it has given uniform success, and he regards it as the ideal method of treatment for necrosis of the nose.

Harris.

A New Contribution to the Treatment of Ozena by Functional Nasal Reeducation.

Robert Foy (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* August 10, 1912) divides the treatment into two stages. The first is a preparatory stage, lasting from eight to fifteen days and consisting, first, of the injection into each nostril night and morning of hydrogen peroxid; second, waiting three minutes before blowing the nose; third, the introduction of boric acid powder in the nostrils by means of an autoinsufflator; fourth, waiting five minutes before blowing the nose; fifth, lavage of the nose with a liter of hot water in which is dissolved a mixture of salt, glycerin and tincture of iodin; sixth, five to ten minutes breathing in the open air; seventh, internal treatment with iodid of potash.

At the end of this time the special treatment is begun, which consists, first, of pulverization with dry air under a pressure increasing from zero up to five kilos; second, massage of the nasal mucosa, especially the head of the middle turbinate and the upper region of the septum with cotton rolled upon an applicator; third, breathing exercises, first with one and after with two nasal olives introduced into the nostrils of the nose, moist air being introduced at intervals. The treatment lasts a half an hour.

During the second stage of the course of treatment, irrigations of iodin are discontinued. Twenty to forty seances are necessary, at first daily, and later every other day. In addition to the treatment with the dry air, three to four times during the day for ten minutes simple respiratory exercises are practiced. These have the object of establishing the nasal respiration and increasing the vitality of the mucous membrane of the upper air passages. Three exercises are given, the first is purely nasal, the second, costodiaphragmatic. At the end of the treatment the patient is usually cured. There can be relapses of a minor degree, which are relieved by reeducation and more careful respiratory and hygienic regime. If the patient is not cured but only benefited, a second course of treatment, lasting three or four months, is in order. Only very rarely is a third course necessary in order to obtain a cure.

At times, after the patient is well, the use of paraffin is desirable but not necessary. The author insists that ozena is not a specific affection, but a trophic disease affecting the passages of the upper air tract and causing a suppression of the functions of this mucosa. Air is the physiologic stimulant of the mucous membrane exposed to the air, just as food is the stimulant of the mucous membrane of the digestive tract. The treatment outlined restores to the nasal mucous membrane its normal physiologic stimulant, air.

The author concludes by giving the report of a number of cases of ozena which have been cured by the method.

Harris.

Fractures of the Nose.

Garel (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* August 31, 1912) dwells upon the importance of the immediate correction of all fractures of the nose, as a means of avoiding extensive operative measures later on.

He reports a case recently under his care where he employed sheets of gutta percha for an external retaining splint, with excellent results.

(Abstractor: As is well known, gutta percha has been used for this purpose in America for a number of years.)

Harris.

The Treatment of Nasal Synechia by Means of Mica Splints.

Moure (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* October 26, 1912). In Moure's hands the thin lamellæ of mica have proved more satisfactory than any other method employed for this condition. He has used it in many severe cases, with excellent results.

It is so thin that it does not cause any irritation and does not interfere with the normal secretions. It is easily sterilized, standing a high degree of temperature. It also permits of being bent. All that is necessary is to give the mica the form that it should have inside of the nose. *Harris.*

The Sphenochoanal Polyps.

Kubo (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie,* March and April, 1913). The result of Kubo's investigations are summed up in the following conclusions:

First, that solitary choanal polyps can proceed in the same way as antrochoanal polyps or maxillary choanal polyps, and have their origin in the sphenoidal sinus, especially in the vicinity of the lower part of the ostium. To these polyps he gives the name of sphenoidal or sphenochoanal polyps.

Second, in the early stages of their formation these polyps are found in the sphenoidal sinus and are discovered accidentally when the sinus is opened, so they are characterized as a polyp-like thickening on the upper choanæ border just below the ostium. This latter phenomenon is an important objective symptom of chronic sphenoidal sinusitis.

Third, in the later stage of their formation, sphenochoanal polyps can become so large that they totally obstruct the choanæ. This is why the differential diagnosis from antrochoanal polyps is very important from a therapeutic standpoint. It is necessary before operation to make a careful examination, because the sphenochoanal polyps can resemble closely the nasopharyngeal fibroma, as the result of their short pedicle and the situation of their base.

Fifth, in Kubo's opinion the majority of polyps spoken of as choanal are in effect sphenochoanal.

Sixth, in sphenochoanal polyps the sphenoidal ostium is generally very large.

Seventh, simple removal with the snare is not sufficient as a radical operation for sphenochoanal polyps. It is necessary at the same time to treat adequately the source of their origin, that is to say, the sphenoidal sinus itself. *Harris.*

III.—PHARYNX.

The Factor Intoxication in the Genesis of Adenoid Vegetation.

FISTRE (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* July 6, 1912). An unusually thoughtful and suggestive contribution to the etiology of adenoids. The article should be read in full, but his conclusions are as follows:

Hyperplasia of the lymphatic tissue of the pharynx constitutes a visible evidence of a defensive action of the infantile organism against a chronic intoxication, whether inherited or acquired.

It forms an integral portion of a complex syndrome, often discrete, i. e., the rachitis, affecting at first the bony marrow, then almost immediately afterwards the lymph organs, and later only the kidneys, spleen and the other internal organs.

In a case where the organism of the infant is profoundly affected, adenoidectomy alone gives indifferent results as regards the later somatic development. To accomplish a complete cure, not merely are appropriate respiratory exercises necessary and a general hygienic and dietetic treatment, but also an actual cure by means of polyglandular opotherapy according to the method of Dupuy. *Harris.*

Preliminary Report Concerning the Passage of Bacteria Through the Tonsillar Tissue as Determined by Experimental Research.

Dr. George B. Wood, Philadelphia (*Transactions American Laryngological Association, 1913*): From a study of our experiments it seems that the following conclusions may be drawn: 1. The tonsils in the hog are more readily infected by the anthrax bacillus than any other portion of the buccal or pharyngeal mucosa. The clinical history of this disease in the hog shows that in the great majority of idiopathic cases the pharynx has been the site of invasion, and in all of these cases of pharyngeal diseases the tonsils are the port of entry. In none of my experiments was there any involvement of the pharyngeal or buccal mucosa other than the tonsils. While the culture of anthrax was generally brought into more intimate contact with one of the tonsils, it was impossible to limit the bacilli to the tonsillar surface, and they came into contact with a large part of the pharynx. In the infection an effort was made to rub the emulsion into one tonsil only and in one case the lesions were limited to one tonsil only, but this was not the tonsil on which the culture had been rubbed. 2. Anthrax bacilli penetrate through the cryptical and not the surface of the epithelium. 3. The anthrax bacillus probably gains access to the parenchyma of the tonsil by passing through the living, unaltered epithelium, and having gained access through the superficial layers of the epithelium, they tend to multiply in the deeper layers and then pass into the interfollicular tissue. 4. The anthrax bacilli penetrating through the living normal epithelium cause a devitalization of the tissue, which paves the way for secondary infection from the staphylococci or other pathogenic organisms. 5. The rapidity of the invasion is influenced both by virulence of the organism and the susceptibility of the individual animal. Following the invasion the subsequent course of the disease is similar to that found in other tissues. The toxin elaborated by the bacilli causes at first an accumulation of polymorphonuclear cells, later necrosis of the tissue cells with disintegration of the nuclei. The germinating follicles show more resistance to the disease than the interfollicular tissue. Associated with the necrotic process is an increase in the number and engorgement of the capillaries, and sometimes there is marked extravasation of the red blood cells. The anthrax bacilli accumulate in the lymph spaces and also around the blood vessel walls. In some of the sections examined the bacilli were found penetrating the blood vessel walls, and a few were found actually in the blood current.

IV.—LARYNX.

Rheumatic Cricoarytenoid Arthritis.

U. Calamide (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie,* May and June, 1912) reports two cases occurring in his own practice. In both the cricoarytenoid articulation was much swollen, reddened and completely immobile during phonation and respiration. The corresponding vocal cord was fixed in adduction, as is found in paralysis of the recurrent. There were evidences of rheumatism elsewhere in the body.

In the first case local and general treatment cleared up completely the laryngeal condition in the course of two weeks. In the second case, a more severe one with pronounced dysphagia, there was a disappearance of the tumefaction and pain, but at the end of two months there had been no change in the condition of the cricoarytenoid articulation and the corresponding vocal cord. *Harris.*

The Treatment of Dysphagia in Laryngeal Tuberculosis.

Combier (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie,* September and October, 1912). After referring to the various older methods employed, the author describes regional anesthesia of the trunk of the superior laryngeal as the most satisfactory means of relieving the difficulty. This can be secured either by the lateral route or the mediate route. The lateral route is the technic which he is wont to employ. A straight needle is employed, attached to a Pravaz syringe. The patient is placed on his back, the head slightly hyperextended, the neck is painted with tincture of iodin, a finger of the left hand which pushes the larynx toward the side to be injected, maps out the interthyrohyoid membrane. Frey advises introducing the needle midway between the superior cornu of the hyoid bone and the posterior superior angle of the thyroid cartilage. One goes in at this level always a little nearer the hyoid bone, at first perpendicularly and then horizontally, from backward, forward, piercing the muscles which offer little resistance, and penetrating a little more than one cm. until we come in contact with the thyrohyoid membrane. At this point the patient should feel a pain in the ear if the nerve is reached. After being satisfied that blood does not follow the needle, the injection is used.

The writer makes use, as most others do, of one cm. of 85 per cent alcohol slightly warmed. The unpleasant results which sometimes follow the use of the alcohol can be avoided by preceding its use with half to one cm. of a weak solution of novocain and adrenalin. The method by the direct line, as recommended by Boncour, has the advantage of permitting a bilateral injection with the same introduction of the needle. There is a risk, however, of breaking the needle, and Combier feels that a second introduction is better. He states that it is rare that the injection is without effect; usually the results are excellent, and it is unattended by any unpleasant effects.

Harris.

Unilateral Affection of the Vocal Cords.

HEYMANN (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie,* January and February, 1913). The usual impression based upon textbook statements for many years is that a unilateral affection of the vocal cords is due to some specific condition. Heymann shows that this is not correct by quoting three cases that Rosenberg has reported showing this condition, which all recovered, and where tuberculosis, syphilis and cancer could be excluded. The fourth case was a case of Herbert Tilleys. Heymann himself has often had an opportunity of seeing isolated affections of one cord, or a part of one cord, due to chronic laryngitis, which persisted for a number of months, or even two years in one case, to finally get well as the result of treatment.

While this is true, he does not dispute the fact that the great majority of affections of the vocal cord have their origin in a constitutional disease. To explain this it is necessary to know exactly in regard to the etiologic conditions.

The exact genesis of catarrhal inflammation is not yet known, but the usual views held would tend to explain why in chronic laryngitis both cords are wont to be involved.

Harris.

The Early Diagnosis of Laryngeal Tuberculosis.

William Lamberson (*New York Medical Journal,* October 19, 1912) has made an examination of the throats of 730 patients who have presented themselves at the tuberculosis clinics of the New York Department of Health. Of these, 244, or one-third, have been found to have tuberculous trouble in the lungs. Of these 244, 45 per cent have had tuberculous laryngitis, and 21 per cent had varying degrees of laryngitis, nontuberculous in origin, making a total of 66 per cent of positive chest cases with some pathologic laryngeal condition.

The author is of the opinion that in many of these cases of pulmonary tuberculosis the laryngeal condition, while apparently catarrhal, is really tubercular. He calls attention to the significance of a unilateral involvement. A cord showing loss of luster, rounded and thickened in parts, slow to respond in adduction, is strongly suggestive of tuberculosis. Later it assumes a bottle or fusiform shape, partly overlapped by a corresponding ventricular band.

The epiglottis in the early stages may show only a slight loss of mobility and change in appearance from the usual pink to a soft, velvety congestion. Later the color may be a deep red. As regards the voice, in his experience it is apt to be weak or husky, rather than hoarse. Incomplete adduction of one or both cords may be due to a lesion in the musculature or to pressure upon the recurrent laryngeal nerve. Pain is not a constant manifestation of tuberculosis of the larynx. Dysphagia is a symptom denoting the involvement of the deeper structures. He rightly recommends that all tuberculous patients should have early and frequent laryngeal examinations. In his experience the majority of cases start with hyperemia rather than anemia. *Harris.*

Thyrotomy for Cancer of the Larynx, With Report of Eleven Cases.

Dr. D. Crosby Greene, Boston (*Transactions American Laryngological Association, 1913*): In a paper presented to this association in 1906 we reported the results of an investigation of the lymphatic drainage of the larynx by means of submucous injections of methylen blue and mercury. The results obtained are confirmatory of those reported by others in showing that the network of lymphatic vessels which extends beneath the mucous membrane throughout the interior of the larynx is richer in the number and size of the vessels in the supraglottic region, relatively poorer in the subglottic portion, while on the vocal cords the vessels are very small and widely separated. These anatomic facts account for the slow growth and late development of the disease in the cervical lymph nodes in cases of epithelioma of the cords, and furnish an argument for the possibility of cure in early cases by the operation of thyrotomy and excision of the growth with a wide margin of healthy tissue. This is supported by the result of the operation in the hands of numerous operators, both in this country and abroad, so that at the present time it is almost universally recognized as the proper procedure for the treatment of early intrinsic cancer of the larynx. Certain details of the technic have an important bearing on the immediate and after-results of the operation. The steps of the operation are: 1. Ether by inhalation, preceded an hour before by ¼ grain of morphin and 1/150 grain of atropin. 2. With the head slightly extended a median incision is made, extending from the lower border of the hyoid bone to the lower border of the cricoid cartilage. This incision is carried down through the prethyroid muscles until the thyroid and cricoid cartilages and cricothyroid membrane have been definitely exposed. 3. A one per cent solution of cocain is injected through the cricothyroid membrane into the cavity of the larynx. 4. The patient is now placed in the Trendelenberg position and a thick pad placed under the shoulders to bring the larynx into prominence. 5. The cricothyroid membrane is next incised in the median line, and through this incision a swab of ten per cent solution of cocain is introduced and applied to the laryngeal mucous membrane. 6. The thyroid cartilage, after a pause of five minutes, is divided from below upwards. In young subjects this may be done with a knife, but in the majority of cases where the cartilage has become ossified, it is best to use strong curved scissors with dull points. 7. The thyroid wings are now widely retracted and an examination of the growth made under good illumination. 8. Beginning at the free margin of the thyroid cartilage, on the affected side in front of the growth, the internal perichondrium is elevated from off the cartilage with a sharp elevator from before backwards to a line well behind the limits of the growth, as well as above and below it. All the soft structures are thus freed from the underlying cartilage. 9. Parallel horizontal incisions are now made with scissors above and below the growth. These incisions are carried about one-half inch back of the posterior limit of the growth. 10. The growth with its surrounding tissue is now entirely removed with a wire snare by which the posterior attachments are severed. Much depends on the proper selection of cases. When the growth is so extensive, even though confined within the cavity of the larynx, that the larynx cannot be opened without cutting into the growth, recurrence is not only possible but probable.

Decannulation and Extubation After Tracheotomy and Intubation Respectively.

Dr. Chevalier Jackson, Pittsburg (*Transactions American Laryngological Association, 1913*): The different forms of laryngeal stenosis associated with difficult decannulation or extubation may be classified into the following types: 1. Pain. Breathing through the neck with a properly placed tracheotomy cannula is much easier than breathing through the mouth, that once the patient becomes accustomed to it for quite a while he does not feel that he is getting enough air through the mouth, even though the larynx is perfectly patulous. In addition to this there is a nervous cell habit arising from previous experience with the stenosis that terrorizes the patient the moment he feels the slightest dyspnea. 2. Spasmodic. This form of stenosis may be associated with panic, or may be excited by subglottic inflammation. It is usually overcome by the same means as those suggested for panic, together with the treatment of inflammatory conditions that may be present. Doubtless one of the chief causes of adductor spasm is the prolonged wearing of the intubation tube. 3. Paralysis. Bilateral ankylosis of the cricoarytenoid joints may prevent decannulation until the laryngeal stenosis is relieved. This operation is not to be advised except in such cases as have remained rigid for a period of twelve months or more, and this is not meant to include the fixation that is associated with malignant, tuberculous or luetic infiltrations. 5. Neoplasms. Decannulation in neoplastic cases will depend upon the nature of the growth and its curability. 6. Hyperplastic. 7. Cicatricial, (a) loss of cartilage; (b) loss of muscular tissue; (c) fibrous. The hyperplastic and cicatricial types of organic stenosis preventing decannulation may be classified as follows: 1. Tuberculosis. 2. Lues. 3. Scleroma. 4. Acute infectious diseases, (a) diphtheria; (b) typhoid fever; (c) scarlatina; (d) measles; (e) whooping cough. 5. Decubitus, (a) cannular; (b) tubal. 6. Trauma, (a) tracheotomic; (b) intubational; (c) operative; (d) suicidal. Conditions outside of the paralytic and neoplastic forms are almost all the result of inflammation, often with ulceration and the secondary tissue changes. In the infective granulomata it is practically always the mixed infections from oral sepsis running that do the harm. The chief exception to this is diphtheria, which is in many cases a distinctly necrotic process. In the rare cases in which laryngeal tuberculosis of such severe type as to require tracheotomy is cured, decannulation presents little difficulty after the infiltrations are reduced. The reduction of these infiltrations by the galvanocautery through the laryngeal speculum is readly accomplished. Should cicatricial stenosis from ulceration remain, it is to be treated in the same way as cicatrices from other causes—by laryngostomy. In those old cases of luetic fibrosis little amenable to the older methods of treatment, salvarsan has accomplished wonders. Dr. Emil Mayer has recommended the use of radiotherapy in the treatment of scleroma. So far, however, the results have been so unsatisfactory that they practically constitute the only cases in which decannulation is impossible. When typhoid fever was prevalent in Pittsburgh, it was found that the ulcerative lesion in the larynx was practically always the result of mixed infection, but in some instances they were due to thrombosis of a small vessel with subsequent necrosis. The after-treatment of these cases is chiefly by prolonged intubation, and in some cases by laryngostomy. Scarlatina may be followed by acute laryngeal stenosis which is cicatricial. Occasionally foreign bodies may ulcerate through from the esophagus into the trachea. A properly fitting tube will not cause any ulceration, if it is free from roughness or sharp edges and is removed sufficiently often to be cleaned. For diphtheria and like conditions I have never seen any improvement on the original O'Dwyer apparatus. When a tracheotomized case reaches the stage when it is to be trained to breathe through the mouth, it is necessary to occlude the cannula. For the reduction of exuberant granulations, nothing has yielded better results than resorcin. As a stimulation to epithelialization the German preparation "scarlet red" (Biebrich) in a sterile 20 per cent mixture has yielded excellent results during laryngostomy. One of the most common causes is the neglect of frequent changes of dressings.

Primary Carcinoma of the Epiglottis, With Report of a Case.

DR. EMIL MAYER, New York, (*Transactions American Laryngological Association, 1913*): Primary cancer of the epiglottis is a rare disease, and up to the present time the treatment has consisted of surgical intervention or the use of radium and the X-ray. I have seen two cases of cancer of the epiglottis. The first case was diagnosed as probable malignant disease of the epiglottis, and the subsequent history was that of primary epithelioma of the larynx with subsequent metastasis, laryngectomy, and death. The second case was that of a man, 64 years of age, who consulted me for some slight difficulty in swallowing, especially the swallowing of cold liquids. His general condition was good. Examination showed some edema of the uvula, and numerous small white spits on the tongue and inner surface of the cheek. The condition was diagnosed as leukoplakia buccalis. He gave no history of lues. After treatment he improved somewhat, but this did not last, and some six or seven months later there was discomfort in swallowing, a nasopharyngeal catarrh, and the white patches still present. There were no other evidences of a diseased condition in the throat. About three weeks later a new condition presented itself on the laryngeal surface of the epiglottis. There was a deep ulceration surrounded by a thickened mass. The pathologist pronounced this cylindrical celled carcinoma. Here I had a case of carcinoma of the epiglottis in its earliest stages. The epiglottis was removed, after the patient had been anesthetized and the suspension laryngoscope placed in position. There was little bleeding at the time of the operation, but twenty-six hours after, the patient began to expectorate large quantities of blood. This bleeding was checked by the application of ice internally and by sprays of peroxid of hydrogen. He left the hospital in ten days. This was the first operation of its kind ever performed under the ingenious suspension laryngoscope of Killian.

V.—MISCELLANEOUS.

Diseases of the Eighth Nerve in Connection With Epidemic Parotitis.

MAUTHNER (*Archiv für Ohrenheilkunde,* Vol. 87, p. 223). The literature on this subject shows that permanent deafness in the affected ear is the result, temporary deafness the exception. A slight disturbance of the auditory nerve and the labyrinth have up to the present time not been described. While the most common period for involvement of the ear in connection with mumps is from the sixth to the fifteenth year, no thoroughly authenticated case has occurred over forty-one. The author makes the following divisions:

1. Cases with permanent abolition of function (deafness or deafness with loss of static irritability).

2. Marked disturbances: (a) With temporary abolition of function; (b) with temporary severe disturbance of function.

3. Slight disturbances for the most part only to be demonstrated by means of the continual tuning fork tests and exact examination of the vestibular apparatus.

1. Cases with permanent abolition of function. Here there is usually no involvement of the middle ear. At times a prodromal tinnitus occurs, usually on the third or fourth day of the disease, when the general symptoms begin to moderate, or during a new onset of fever with a secondary involvement of the glands near the ear. This may occasionally take place during convalescence as late as the fifteenth day. It is wont to be unilateral. The noise is of all degrees of severity from the slightest to the most intense. It is followed in the briefest interval of time by complete unilateral deafness. Bilateral deafness is also occasionally met with. In seventeen cases described by Fournies, nine were bilateral.

The characterization of apoplectic form seizure Mauthner does not approve of, inasmuch as the onset, while rapid, is by no means always sudden, there being cases on record where complete deafness has not resulted until at the end of five weeks. The disturbances of the vestibular apparatus can be of all degrees, from complete loss to entire preservation. The functional tests of two cases are given, illustrating the extremes. Vertigo is in the majority of cases associated from the beginning with the noises in the ear, separated only by an interval of hours, and less often of days. It is wont to disappear in the course of several days. At times it can be of such an intensity as to lead to vomiting, and the patient is unable to go around. Slight degrees of vertigo will be apt not to be discovered.

2. Marked disturbances associated with either (a) temporary abolition of function, or (b) temporary severe involvements of function. The clinical picture of these cases in their onset and course does not differ from those in the first group. The author reports a case of complete loss of hearing which returned after eight days, and a second case where there was serious impairment, also restored in the course of eighteen days. Cases belonging to this group are not common.

3. Slight disturbances. For the most part only to be recognized by the accurate testing with tuning forks and the exact examination of the vestibular apparatus. The discovery of this group of cases is the result of the recent more accurate methods of testing the vestibular apparatus. The author recognizes the difficulty of finding such cases. In the course of two years he has had an opportunity of examining a hundred cases of parotitis, of different ages and both sexes. Of these one hundred cases, twenty-one were observed during the first four weeks of the attack of mumps. Of the hundred cases, in four, disturbances of the ear were shown which had a direct relation to the attack of mumps. The functional tests of these four cases are given in detail. The disturbances in question in three of the four cases had to do with the cochlear apparatus. In the fourth case, hearing for both the sound and the speech were involved. The impairment in all cases was found in the middle register between "c" and "a." With the exception of the fourth case, it was only slight, representing about 10 per cent, but repeated tests established its accuracy.

The author discusses at considerable length the views of Wittmaack in regard to a toxic involvement of the acoustic nerve, and reaches as the result of his studies the following conclusion as regards the pathology of the ear complication of mumps:

The clinical signs in all three groups resemble for the most part the picture of acute toxic and infectious neuritis of the eighth nerve. The slight disturbances (group 3), which are here described for the first time, will tend to expand the limits of the clinical picture and cause us to suspect analogous disturbances of other acute injuries. *Harris.*

Poisoning From the Use of Menthol.

LEROUX (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* September 11, 1912) calls attention to the great danger of using menthol in concentrated form, or even in any strength, in the respiratory tracts of young infants. He reports a number of cases where asphyxia and almost death have occurred. This may be due to the (a) falling of the menthol oil in the larynx, (b) spasm of the glottis due to the contact of the oil in the larynx, (c) hypersecretion which is produced in the nasopharynx by the presence of the drug, (d) reflex irritation upon the lungs and heart as a result of the effect of the drug upon the nasomucosa.

He strongly urges that the drug should never be used in infants under three years of age, great care in children up to the age of seven, and in adults the employment of a dose not stronger than 10 cm. to 50 dr.

Apart from asphyxia, acute conjunctivitis, laryngeal cough, pseudoerysipelas of the face and noises in the ear have been met with as the result of the use of menthol, while in certain cases its continual employment produces an erythema of the lips and of the nose, and a condition akin to morphinism and cocainism, which may be termed mentholism. *Harris.*

The Diagnosis and Treatment of Cicatricial Stenosis of the Esophagus.

GUISEZ (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie,* Vols. XXXIV, XXXV, 1912) in 1200 esophageal examinations has met with 90 cases of cicatricial stenosis, making it, next to cancer, the most frequent condition met with in the esophagus. In addition to the usual causes for stenosis, he has met with cicatricial stenosis of inflammatory origin in a number of cases. Here there has been no wound or traumatism, but traces of subacute or chronic esophagitis which has little by little produced an inflammation and sclerosis of the walls. In the chronic form it is due to chronic irritations, most often alcohol, or the esophagus may be incompletely compressed by a growth from without. Most frequent of all the causes, however, is spasm, usually the result of improper mastication.

Guisez lays much importance upon the previous history. Among physical signs he speaks of inspection, palpation and auscultation. Catheterization is condemned. The three modern methods are radioscopy, radiography and especially esophagoscopy. Catheterization with a soft bougie he advises to precede the endoscopy, in order to determine the probable situation of the stenosis. He is in the habit of making the examination in the Rose position, and finds local anesthesia sufficient except in children.

Where the stenosis is due to a foreign body, simple ulceration or an esophagitis, it is usually single. Stenosis due to caustics is almost always multiple. The usual site of the stenosis is either at the beginning of the esophagus or at the cardiac end. Stenosis of the cardia is most frequent of all, 33 to 40 per cent. In the diagnosis of the esophageal stenosis, spastic contractions are to be borne in mind. Occasionally also congenital stenosis will be met with. While the history is of value, it sometimes is misleading. Patients will give a definite cause to the stenosis when the condition may be of a malignant nature, for example. As complications he mentions obstruction from the bolus of bismuth which had been swallowed in connection with the radioscopic examination. The inflammation of the mucosa can go on to a phlegmonous condition, localized or diffuse. Where the mediastinum is involved, death is wont to occur very rapidly. The prognosis of cicatricial stenosis is bad. Billroth in a series of cases from 1877 to 1886 reported a mortality of 38 per cent. It may be stated that abandoned to itself, a cicatricial stenosis is fatal sooner or later. The prognosis depends to a considerable degree upon the age of the patient.

The treatment has two objects in view: the dilatation of the stricture sufficient to permit nourishment being swallowed, and second, to effect alterations in the cicatricial tissue of the walls of the esophagus. Dilatation by means of the esophagoscope is wont to produce much better results than blind dilatation. Where the stricture yields easily, it is possible to rapidly increase up to No. 18 or 20 at the first sitting. In cases where it is more dense, it cannot be possible to use the bougie higher than No. 13. In adults, 20 mm., in children under six years, 6 mm., and from six to fifteen, 16 mm. should be the maximum of dilatation.

In two particularly difficult cases, Guisez has succeeded by means of the Plumer thread. In certain cases he has employed with advantage the Laminaria dilatation as recommended by Ebstein; also, that of v. Acker, which consists in a drain, by means of a stiff bougie left in position. His best results have been obtained, however, by dilator drains of rubber or metal in the shape of a collar button and retained in position by means of a silk thread fastened at the upper end. Esophageal dilatation should not be undertaken for a number of weeks after the burn, or until all the acute inflammation has subsided. When the dilatation is easy, the introduction of the bougie with the esophagoscope can be abandoned in favor of simple dilatation. The patient should not be allowed to dilate his own strictures until it is certain that the introduction of the bougie can do no harm. The author has used internal esophagotomy for many years with success and has a special instrument for that purpose. More recently, however, he has abandoned his procedure in favor of electrolysis, for which he has devised a set of electrodes with which he has had excellent results. *Harris.*

Lactic Ferment in Rhinology.

SUNE Y MEDAN (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie,* January and February, 1913) has employed the lactic ferment, either in the form of powder or solution, in various affections of the nose. In acute rhinitis the attack can be decidedly shortened by the use of this remedy. He is in the habit here of making use of the powder, introduced, if necessary, after the turbinals have been shrunkened with a weak solution of cocain. The canula should be carried high up into the nose. The insufflation will have to be repeated six to eight times during the day. The results are uniformly good.

In catarrh of the nasopharynx the pulverized ferment is applied through the mouth, the canula being carried in behind the palate. In certain cases, in addition, the nose is to be painted with the liquid lactovacilline and immediately afterwards the powdered ferment insufflated into the nose. The results here are also good. The viscosity and the bad odor are wont to disappear and the patients find themselves much relieved.

In ozena he pursues the following technic: Cleansing of the nose, removing of the crusts, if necessary; second, the large insufflation of the lactovacilline powder; in certain cases, in place of the powder, liquid vacilline has been used. The patient returns every week or every two weeks for observation. He states that he can truthfully say he has never seen a case of atrophic rhinitis which has not been more or less benefited by the treatment. The advantages of the treatment are its ease of application and the absence of all irritating effects. The patient is able to make the applications at home. It is possible in 40 per cent of the cases to show that a cure has taken place in from four to eight months. By cure is meant a disappearance of the crusts and of the odor.

Harris.

Further Observations on Some Anatomic and Clinical Relations of the Sphenoid Sinus to the Cavernous Sinus and the Third, Fourth, Fifth, Sixth and Vidian Nerves.

DR. GREENFIELD SLUDER, St. Louis (*Transactions American Laryngological Association, 1913*): In a previous communication (last May), in which the question was raised as to the etiology and treatment of migrain, Sluder made the statement that he believed that many (but not all) of the recurrent headaches which bear the name of migrain are sphenoidal empyemata which have lost most, if not all, local signs, or which were started as such empyemata, and that the nerve trunks had become involved either by extension of the inflammation (or its toxin) through the thin wall separating the sphenoid sinus from the adjacent nerve trunks. The results obtained during the past year in a goodly number of cases strengthen his belief in the correctness of the deductions detailed in that report.

Sluder observed that the third, fourth, and three divisions of the fifth, sixth and Vidian frequently lie in close association to the sphenoid sinus, a deduction drawn from specimens which he studied by cross sections. His findings, with the exception of the Vidian nerve, were corroborated by Ladislaus Onodi (*Archiv. fuer Laryngologie,* Bd. VI, Heft II, July 10, 1912). This author's method was to follow the nerve trunks in certain specimens, sometimes to remove the wall of the sphenoidal sinus, and then to study the relations of the nerve trunks thereby exposed. He found that they were in these close associations for varying distances, sometimes even as much as 20 mm.

He did not consider the cavernous sinus in these relationships. He pictures specimens where the sphenoid sinus extended so close to the clivus of Blumenbach as to make transparency of the separating bone, and shows how this brings the sixth into these associations. From an inspection of Dr. Warren B. Davis' 145 Caucasian specimens showing the nose and accessory sinuses from the eighth week of fetal life to the twenty-fifth year uninterrupted (several specimens for each year, except the eleventh year), Sluder observed that the sphenoid sinus spreads laterally at an early age, reaching to close proximity to the second division of the fifth as early as two and one-half years, and that this condition runs almost constant throughout the series. Its development (Davis) begins in the anterolateral aspect of the body, and slowly extends backward, spreading, however, rapidly laterally to approach the foramen rotundum, and then proceeding backward. As early as the sixth year the Vidian canal may be approached. Sluder considers that if he is right in the conclusion that the mode of production of these headaches—the pathologic sequence—is the close association of the sphenoid sinus to the nerve trunks, and that the inflammatory processes are transmitted through the thin bone separating the cavity of the sphenoid from the associated nerve trunks, then it is necessary that such anatomic associations be formed in early life as an explanation of such headaches beginning in early life. Another year of clinical observation strengthens Sluder's belief that the pathologic process underlying these cases is a hyperplastic sphenoiditis. From an observation of 100 cases he concludes that the second division of the fifth and Vidian are the nerves most frequently involved (95 per cent). They may be involved singly, or together, then making the picture which would otherwise emanate from the sphenopalatine ganglion. It is difficult to differentiate this class of cases from sphenopalatine ganglion neuralgia; hence, one should be carefully on guard. Since the previous communication, three medicines have been tried for intrasphenoidal use: Iodid of potassium in water, 2 to 5 per cent, proved to possess nothing upon which its use may be recommended; it is apparently inert. One per cent chlorotone in water proved to lack recommendations. The proprietary "cresatin" also failed to prove better adapted for these purposes than carbolic acid or oil of wintergreen. The medicines which have so far proved of greatest benefit are: One per cent carbolic acid in oil; 2 to 10 per cent oil of wintergreen and aqueous solutions of sodium salicylate, 2 to 5 per cent. These have been successful in allaying the pain long after the sinus was satisfactorily opened and the wound healed.

Foreign Bodies in the Esophagus, With Report of Two Cases.

Dr. Cornelius G. Coakley, New York (*Transactions American Laryngological Association, 1913*): The first patient, a boy 16 years of age, thought he had swallowed a piece of plate in his soup. He had a temperature of 105,° rapid pulse and pneumonic area in the right lung, when first seen. Examination of the pharynx showed edema extending from the vault of the nasopharynx as far down as one could see with the laryngeal mirror, or feel with the fingers. It much resembled a retropharyngeal abscess. The X-ray showed a triangular shaped foreign body. It was jagged and evidently had cut into the mucous membrane so as rapidly to infect the pharyngeal mucosa, and the secretion from the infected pharynx passing into the larynx, setting up a septic pneumonia within twenty-four hours. The boy died fourteen hours after the operation, from acute septic pneumonia. This case shows the necessity for the prompt operative relief for removal of sharp foreign bodies. The same day I saw a boy three years of age who had swallowed a coin. The X-ray showed the coin at the level of the sixth cervical vertebra. The child had no symptoms. The coin was removed by operation and the child suffered no subsequent discomfort. In this instance the coin had been in position for five days before it was removed.

Phlegmons of the Upper Respiratory Tract.

Dr. John O. Roe, Rochester (*Transactions American Laryngological Association,* 1913): The phlegmon is simply another name for inflammation, although clinically it is regarded as an extraordinarily intense inflammation and looked upon as being of more or less malignant character. We may have an inflammation of this character in any part of the body, the intensity of which may depend upon the virulence of the infection and the resistance or lack of resistance of the patient, or of the tissues, to such infection. Owing to the difficult breathing or choking which such inflammation causes in the upper air passages, it has received the name of angina. Inflammations of a severe grade, termed phlegmons, may involve any portion of the upper respiratory tract. We almost invariably find a phlegmon attacking one whose general condition has become weakened. The treatment of these infections requires no special law unto itself, and must be dealt with according to the location and associated complications.

Nitrous Oxid Gas, Essence of Orange, Ether and Sequestration in General Anesthesia for Operation in the Upright Position.

Dr. Thomas R. French, Brooklyn (*Transactions American Laryngological Association, 1913*): We are now able to state that as a result of the movements of the new operating table, and also as a result of the tests made in our clinic at the Long Island Hospital, that operative work in the upright position can be done with still less loss of blood, with the need of still less of the anesthetic, and with less disagreeable conditions during recovery from the anesthetic. Sudden changes in posture or sudden disturbances of the body while the patient is under a general anesthetic, predisposes to shock, the effects of which are manifested both during the operation and during the recovery from anesthesia. After a careful study of this phenomenon, I became impressed with the value of elevation of the body from the recumbent position to the upright position without jarring, and this led to the construction of the chair table. By the aid of a new attachment all the movements of the body are now made with as great freedom from jarring and disturbance of balance, with infants and very small children, as had hitherto been possible only with adults or larger children. The resulting improvement in conditions leaves no doubt of the accuracy of the observation. One of the most important and valuable recent additions to methods of anesthesia is the ability to omit or bridge the second stage, or stage of excitement, and we have been impressed with the desirability of attaining narcosis without struggle. This can be accomplished with nitrous oxid gas, but with greater ease and certainty with the essence of orange. The discoverer of the remarkable effects of the essence of orange as a preliminary to ether, Dr. Gwathmey, of New York, has demonstrated the possibilities and has proven to our satisfaction that it greatly assists in the reduction of shock by bridging the stage of excitement. Anesthesia conducted in this way up to the time of raising the body to the upright position is a contributory factor in reducing hemorrhage and also in reducing the quantity of anesthetic and in shortening and modifying the anesthetic after-effects. Irrespective of all other conditions, there is a well-defined relationship between the degree of skill in which a patient is anesthetized in the upright position and the amount of hemorrhage is reduced if the anesthetic is smoothly given. A preliminary use of nitrous oxid gas or the essence of orange and the drop method of anesthol, followed by the drop method of ether with the open inhaler, and later with the Allis inhaler, will insure a prolongation of the anesthesia after the body has reached the upright position, a reduction of hemorrhage, and a reduction in disagreeable sequelæ. Sequestration of the limbs produces a congestive hyperemia, and is produced by means of inflated blood pressure cuffs about the thighs and arms; this reduces the amount of blood in the head. This, in association with the upright position which has been carried out in fifty-eight cases during the past season, reduces still further the amount of anesthetic required and still further the loss of blood. There need be no apprehension of subsequent bleeding.

History of a Broncholith, Bronchial Calculus, or Lung Stone.

DR. WALTER F. CHAPPELL, New York, (*Transactions American Laryngological Association, 1913*): This case is cited to show that all causes of asthma are not centered in the nose, nor all throat coughs in the larynx. The patient was a woman, 52 years of age, who was attacked by severe wheezing, the spasmodic cough lasting an hour at a time, and by intense tickling in the throat. The tickling was below the larynx, and the wheezing in the upper part of the chest, in front and behind. The patient soon raised a small calcareous mass, but the asthma continued. Another coughing spell resulted in the expulsion of a large calcareous mass. Immediately all wheezing stopped and the breathing became absolutely free. Later another calcareous mass was coughed up. Chemically these masses consisted of phosphate of lime. The patient gave no history that would give a clue to the cause for the existence of this condition.

The Removal From the Esophagus of a Plate of False Teeth Embedded for Eighteen Years, by Means of the Esophagoscope.

DR. D. BRADEN KYLE, Philadelphia (*Transactions American Laryngological Association, 1913*): This foreign body which I present was very difficult to locate, in spite of its size and shape, on account of the granulation tissue which had organized into fibrous tissue, together with the curvature of the spine, as the X-ray picture shows. The patient swallowed the upper suction plate with four front teeth attached while sleeping. The plate lodged in his throat, and a physician who was called in wished to push it down into the stomach, to which the patient objected. He then went to a hospital where, after the passage of several instruments, he was assured that he had never swallowed the plate or that it was no longer in the esophagus. For a few weeks after the patient felt slight pain in the lower part of the neck at the point he always felt that the object had lodged. After that time there was no sensation, but swallowing has always been difficult. The Kahler esophagoscope was used, and many attempts were made before this foreign body was successfully removed. By loosening the plate from the fibrous bed by setting up slight inflammatory action and then waiting a few days, the plate was removed without much ulceration of the structure.

ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY.

Vol. XXII. DECEMBER, 1913. No. 4.

LVI.

HISTOLOGIC PATHOLOGY OF THE ACCESSORY SINUSES.*

By Joseph C. Beck, M. D.,

Chicago.

In presenting this subject, it is the purpose of the writer to illustrate histologic specimens of tissues removed from cases operated upon for the relief of chronic sinusitis and to draw some practical conclusions as to the etiology and treatment of these affections. It is intended to bring out most prominently the minute pathology in cases of chronic sinusitis, suppurative and nonsuppurative, in which the symptoms of asthma were prominent.

The work is based upon the examination of a large number of specimens from one hundred and forty cases operated upon for chronic sinusitis, and typical specimens have been selected for this publication. The clinical data or histories, course and final outcome of these cases, would aid a great deal in this report, but for lack of time and space they will be omitted. Suffice it to say that the study of minute pathology has brought a wealth of information to the writer, and the study of these specimens, in connection with the clinical phase of the subject alone, is of considerable practical value.

---

*Read before the American Laryngological, Rhinological and Otological Society, Washington, D. C., May 8, 9 and 10, 1913.

In the study of microscopic specimens of middle turbinates and ethmoid curettements of the cases of asthma, whether in suppurative or nonsuppurative cases, it has been noted that the bone changes are quite uniform in showing the formation of large areas of rarefaction. (Figs. 7, 15 and 16.) However, we have very little evidence of inflammatory processes about these rarefied areas. The tissue covering the bone, nevertheless, shows pathologically considerable variation. In some cases there is a great deal of inflammatory edema, with very little inflammation and round cell infiltration. (Fig. 3.) In others the round cell infiltration and old connective tissue predominate (Figs. 4 and 6), while in still other cases the glands are distended and increased in number (Figs. 2 and 3), or the glandular structures have undergone a myxomatous degeneration. (Fig. 3.) The epithelium varies considerably in its structure; at times it is very markedly thickened columnar (Fig. 14), as well as stratified. (Fig. 13.) Cilia can very seldom be discerned, and in some cases there are areas where the epithelium is entirely wanting.

In regard to the microscopic examination of cysts in the middle turbinates, they resemble considerably the changes as mentioned above, except that the lining epithelium of the cavity and the subepithelial structure appear to have characteristic differentiation. The epithelial cells are arranged in regular columns with nuclei at the base. The basement membrane is very thick and appears to form the endosteum of the cysts. The contents of the cysts are homogeneous in character. (Fig. 8.)

In the chronic nonsuppurative forms of sinusitis, where the symptoms of asthma are not present, the minute pathology appears to differ in one main point, in that the rarefying osteal change is not as prominent; and the writer wishes to call attention to this for the purpose of throwing some probable light on the etiology of the asthmatic symptom. The structure covering the bone has about the same type of change as in the asthma cases; that is, the absence of the round cell infiltration.

The inflammatory edema and secondary myxomatous change are very frequently found at the expense of degenerative changes of the mucous glands. In many of the sections one can make out the beginning of polypus formation. Of course, there are quite a number of sections encountered in which a mixture of inflammatory as well as noninflammatory changes is found, as will be shown in connection with the description of the suppurative type of chronic sinusitis.

In reference to hay fever, it has been argued by many that the sinuses are perhaps the seat of the pathologic change, and treatment directed to them has been reported of value, especially the removal of middle turbinates and curettage of the ethmoid, also washing out and treatment of the cavities, as the antrum, frontal and sphenoid. In operating on a number of these cases and examining the tissues histologically, I have found nothing markedly changed in the middle turbinates. The mucous glands seemed perhaps to be in the state of hypertrophy. (Fig. 5.) Of course, the operations were always done long after or preceding an attack.

In regard to the chronic sinusitis of the suppurative form, we find that the principal change in the early stages is that of marked round cell infiltration, especially in the subepithelial structure (Fig. 12), and the glands appear to be distended in areas, while, in others, destroyed by the inflammatory change. The epithelium is very frequently thickened and inspissated with pus (Fig. 12A), and the bone is seldom changed.

Later, however, the connective tissue fibrosis is found quite prominent, and in many instances the bone is also infiltrated with round cells. (Fig. 12B.) If the process has been of long standing, areas of necrosis in the bone will often be found, especially in curetted portions of the ethmoid. (Fig. 12C.) In connection with this necrotic process distinct areas of granulation tissue are encountered, and in some instances necrotic bone in the form of small sequestra.

In regard to the lining of the cavities, as the antrum, frontals and sphenoids, there is found in the long protracted cases a marked thickening with no evidence of any glandular structures, and many areas of myxomatous degeneration (Fig. 12D); again, a great deal of round cell infiltration and a thin layer of flattened epithelium. (Fig. 12E.) This latter condition is, however, not constant, for at times the epithelium is markedly thickened. (Fig. 12F.) The importance of the epithelium, however, is self-evident, in that its presence prevents spontaneous obliteration of the cavities, and its complete eradication is necessary in operative procedures in order to obtain this result.

In reference to the surgical pathology of polypi, we have found the most frequent condition to be a fibrosis with areas of myxomatous degeneration and inflammatory edema. The round cells are not very prominent except near the surface. (Fig. 12G.) The epithelium covering the polypi will depend on whether there is present pus or secondary recurrence after operative procedures. In the latter condition, the epithelium is markedly thickened (Fig. 12I); in massive polypi, which are crowded within the nares, also in the multiform polyposes, in which the nares are distended by polypi, the epithelium covering is thick (Fig. 12H). As to solitary polypi, especially those of the sphenoid, we have noted a cyst formation. (Fig. 12I.) These cysts are not at all uncommon in the polypi of the nonsuppurative form of sinusitis.

In assuming that atrophic rhinitis belongs to the group of sinus involvement, either primary or secondary, we find the pathologic changes in the middle turbinate to be as follows:

In the early stages the bone has many areas of rarefaction. The soft structure shows areas of marked degeneration in the glands, and the epithelium covering the turbinate exhibits early tendencies to metaplasia or hornification. (Fig. 1.) Later, the bone shows a disposition to shrink, due to the preponderance of fibrous connective tissue. The glands practically are all destroyed, and the epithelium has still greater metaplastic change with many areas of denudation. (Fig. 10.) Similar changes are found in the curetted portions of the ethmoid in atrophic rhinitis.

CONCLUSIONS.

First.—The pathologic changes found in the middle turbinates and curetted portions of the ethmoid in asthma cases and in nonsuppurative sinusitis are very striking in that the bone shows rarefaction somewhat resembling that found in early bony changes of osteomalacia, acromegalia and otosclerosis. This pathologic finding is suggestive of a possible etiologic factor in some disturbances of the polyglandular system or the glands of internal secretion.

Second.—In the nonsuppurative sinusitis the conspicuous absence of inflammatory elements, as the round cell infiltration. and the presence of inflammatory edema or myxomatous degeneration at the expense of loss of glandular structures, is very apparent.

Third.—In the suppurative forms of sinusitis the great prevalence of round cell infiltration with tendencies to necrosis and granulation formation is very characteristic.

Fourth.—The lining of the larger sinuses, antrum, frontal and sphenoid, in the markedly protracted suppurative types, is so changed as to preclude resolution, and no matter how well these cavities may be drained or ventilated, their obliteration or eradication cannot be brought about unless the entire epithelial lining is destroyed.

Fifth.—The pathologic characteristics of both suppurative and nonsuppurative forms of sinusitis are very frequently met with in combination in the same case.

Sixth.—The great similarity in the changes of atrophic rhinitis in the early stages and of nonsuppurative sinusitis, especially in the bone, would suggest a similar etiologic factor in the disturbances of the glands of internal secretion, as mentioned in **first conclusion.**

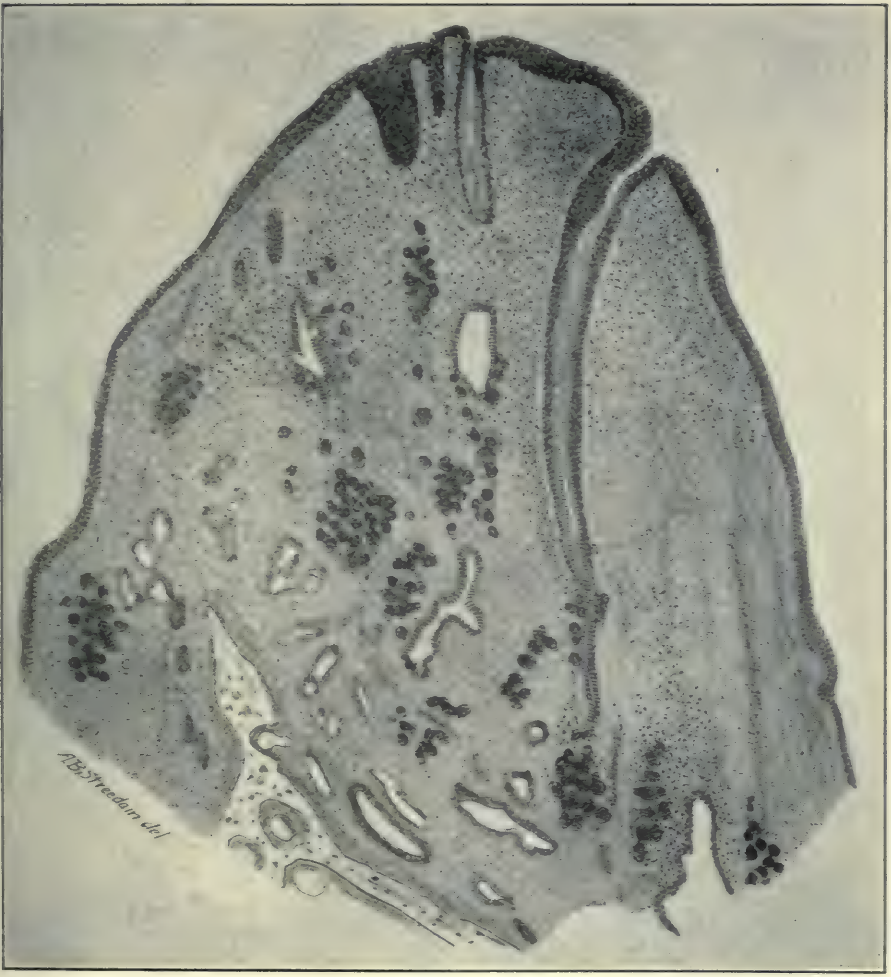


FIGURE 1.

Middle turbinate. Atrophic rhinitis.

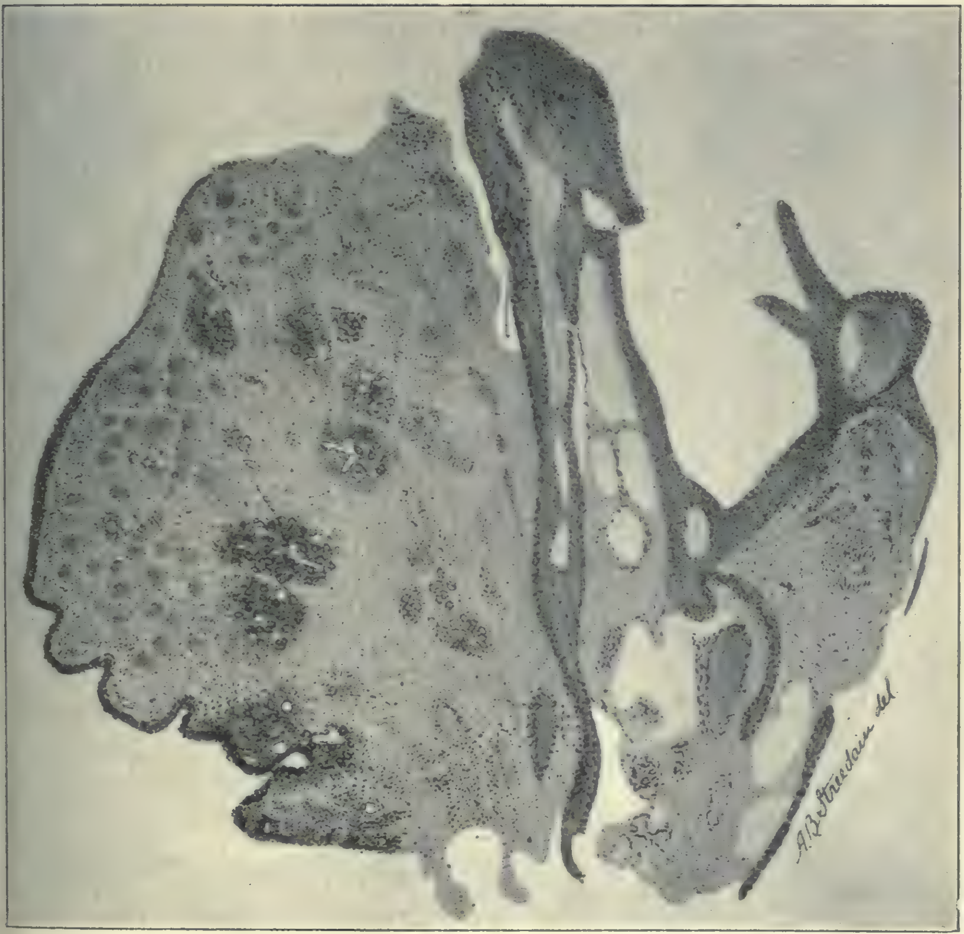


FIGURE 2.

Middle turbinate. Nonsuppurative sinusitis. (Asthma.)

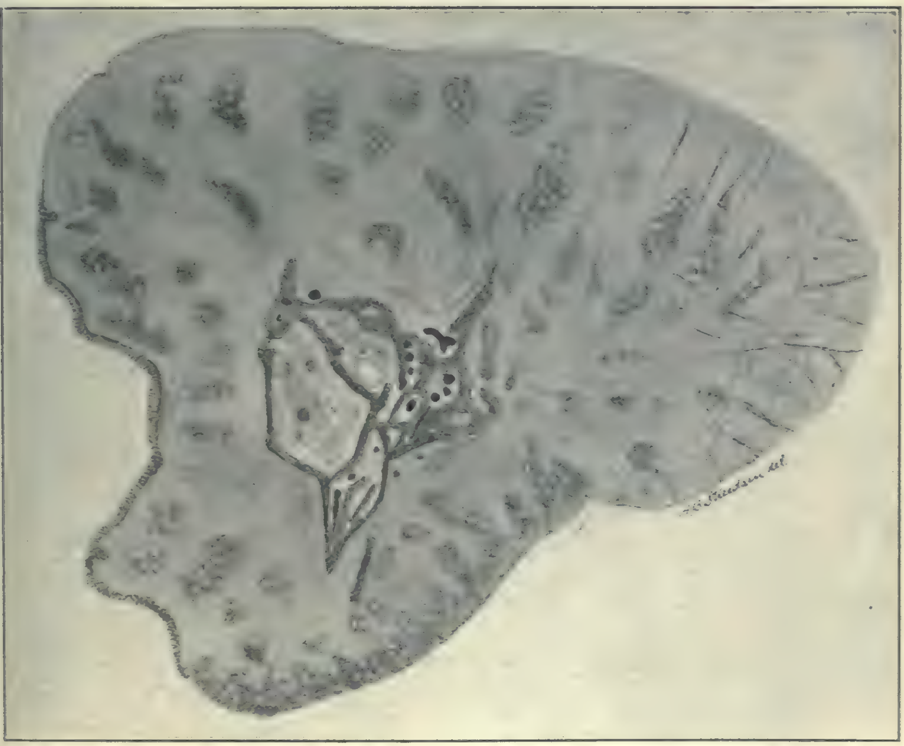


FIGURE 3.

Middle turbinate. Nonsuppurative sinusitis. (Asthma.)

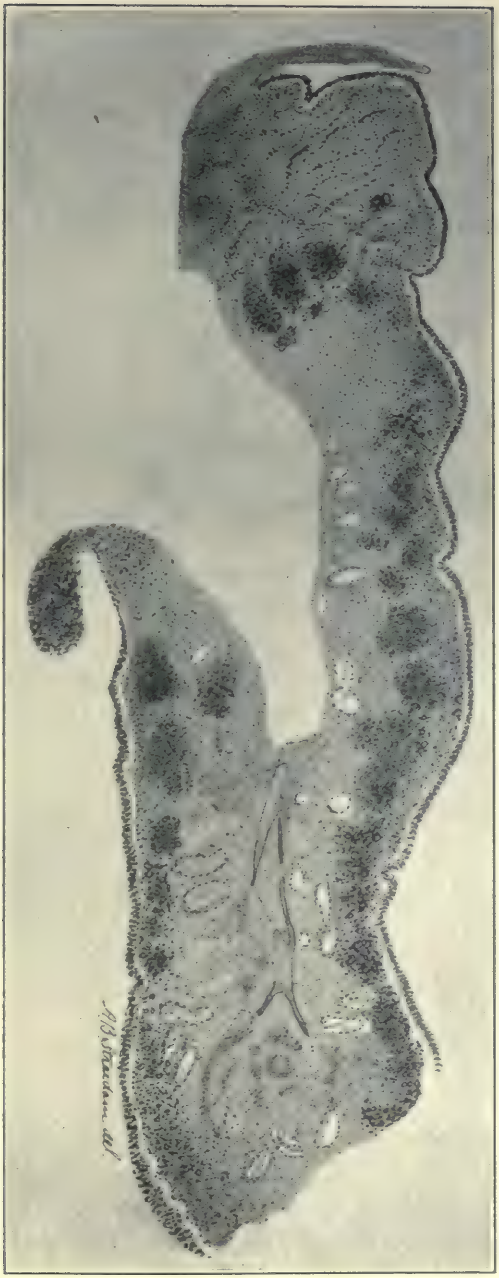


FIGURE 4.

Middle turbinate. Nonsuppurative sinusitis.
(Asthma.)

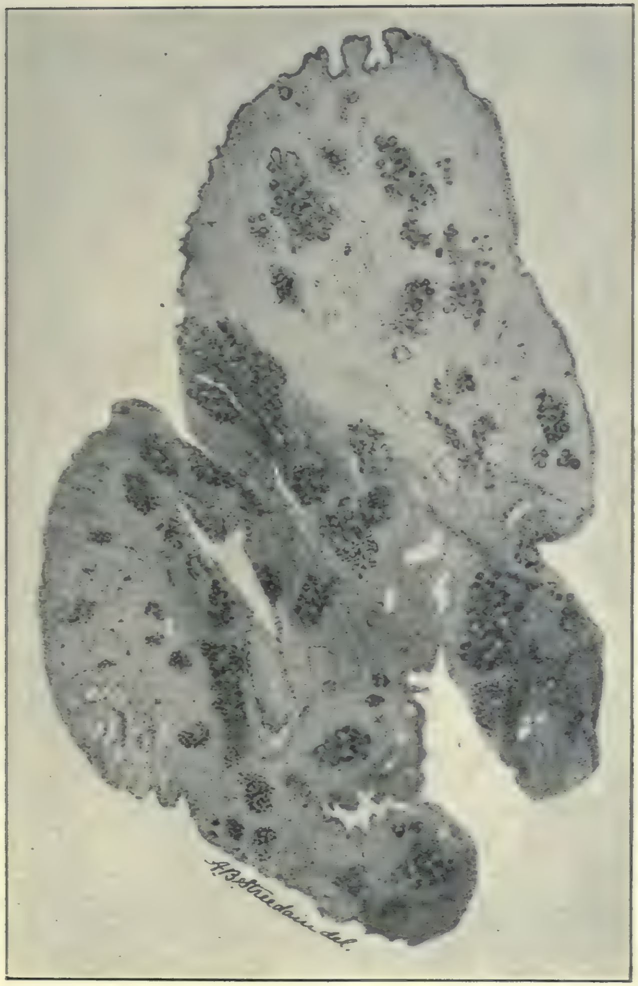


FIGURE 5.

Middle turbinate. Hay fever. (Asthma.)

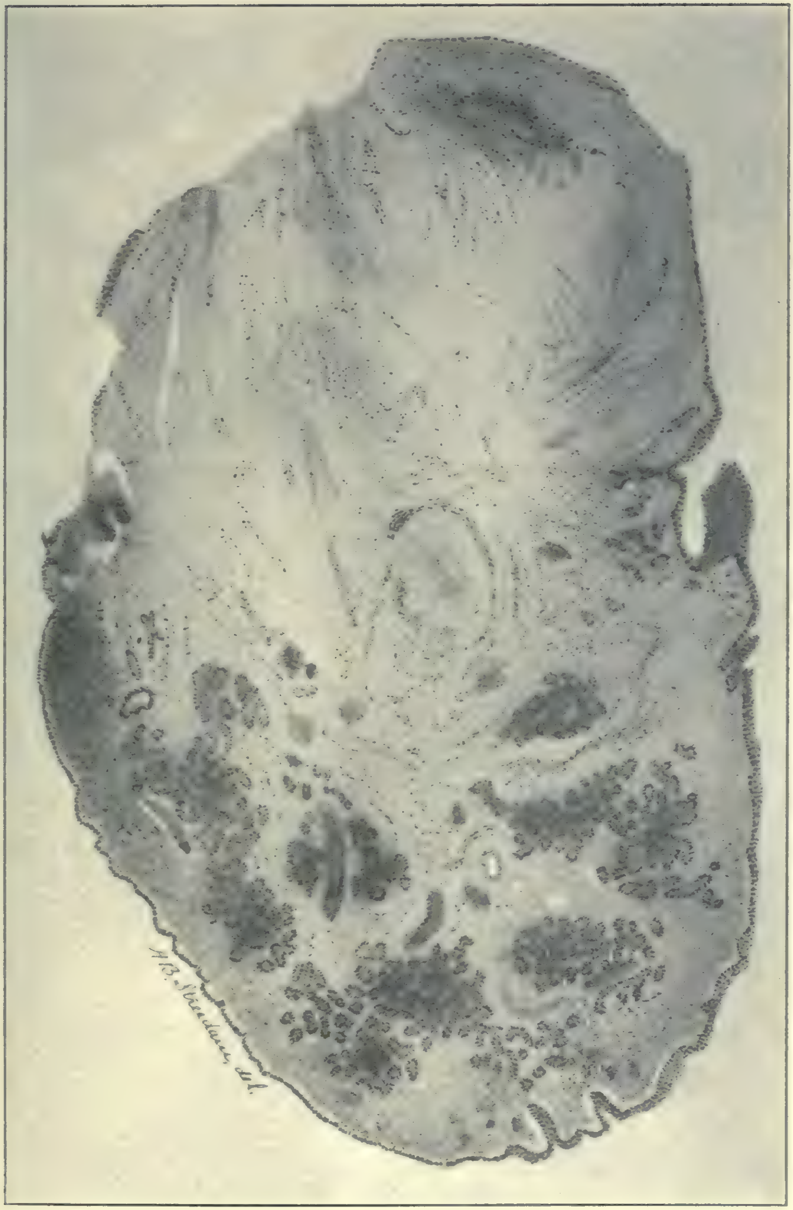


FIGURE 6.

Middle turbinate. Nonsuppurative sinusitis. (Asthma.)

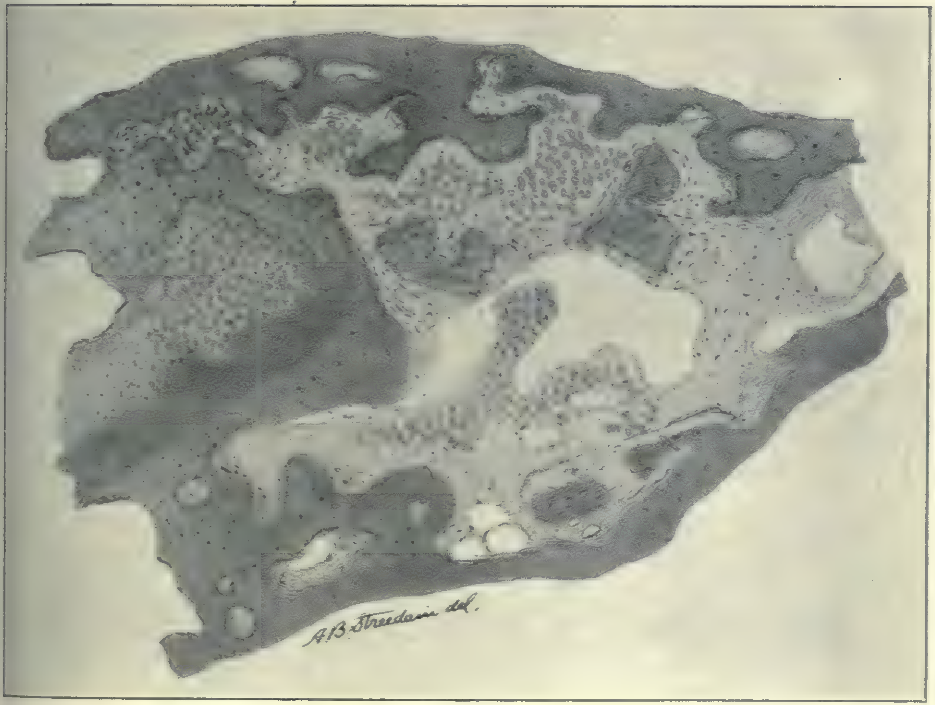


FIGURE 7.

Middle turbinate. Nonsuppurative. (Asthma.)

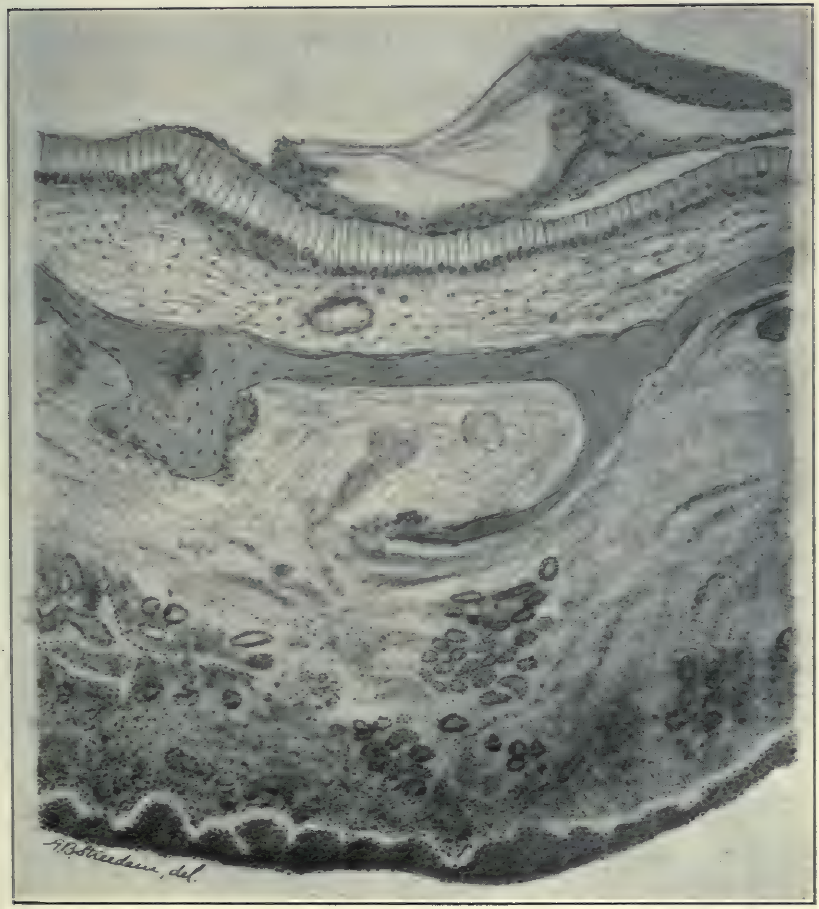


FIGURE 8.

Middle turbinate. Cystic formation. Nonsuppurative sinusitis.
(Asthma.)

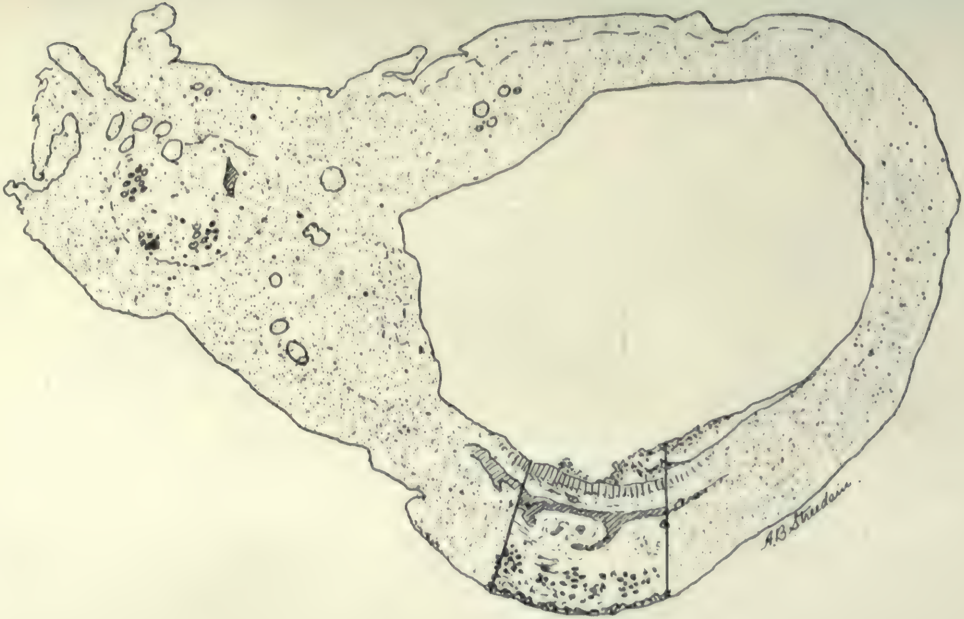


FIGURE 9.

Key to Figure 8.

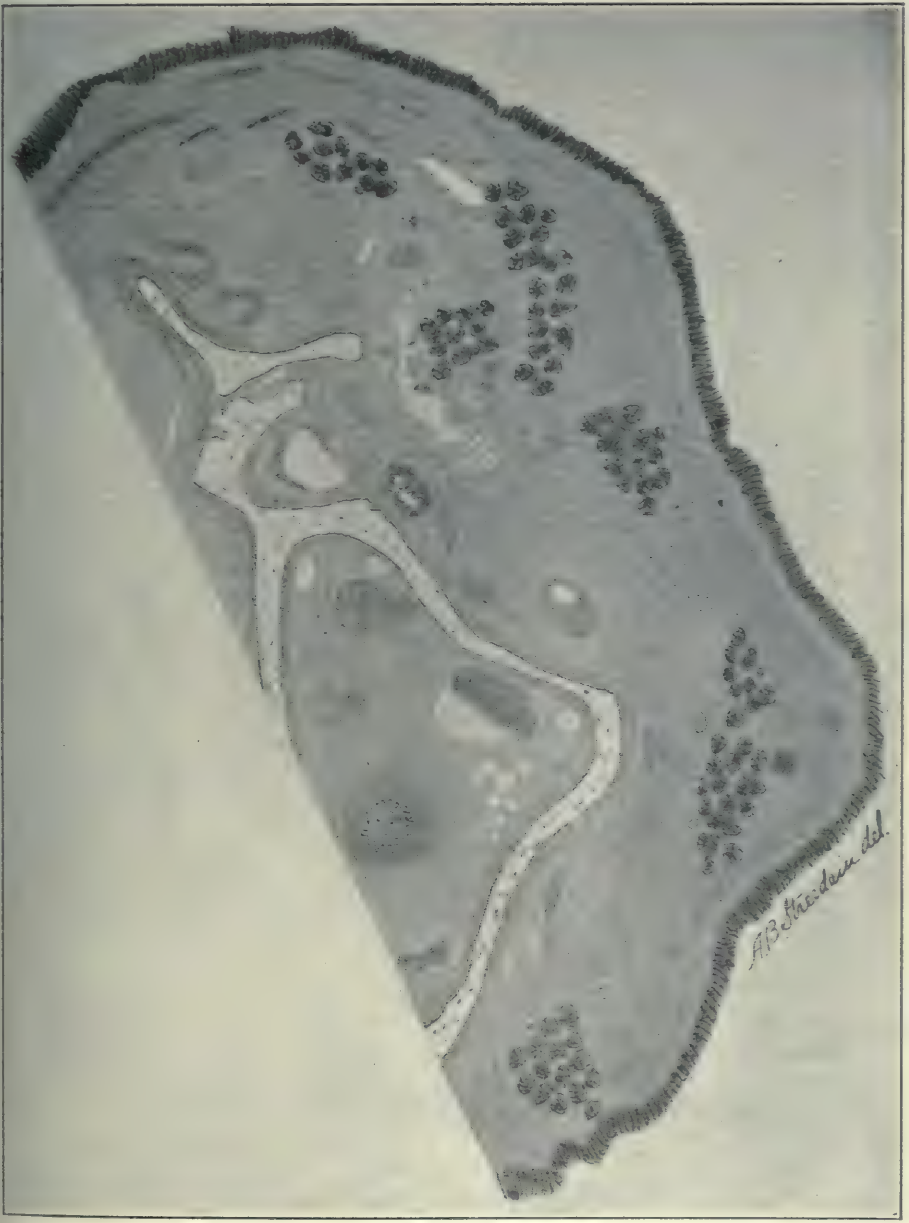


FIGURE 10.

Middle turbinate. Atrophic rhinitis. (Late stage.)

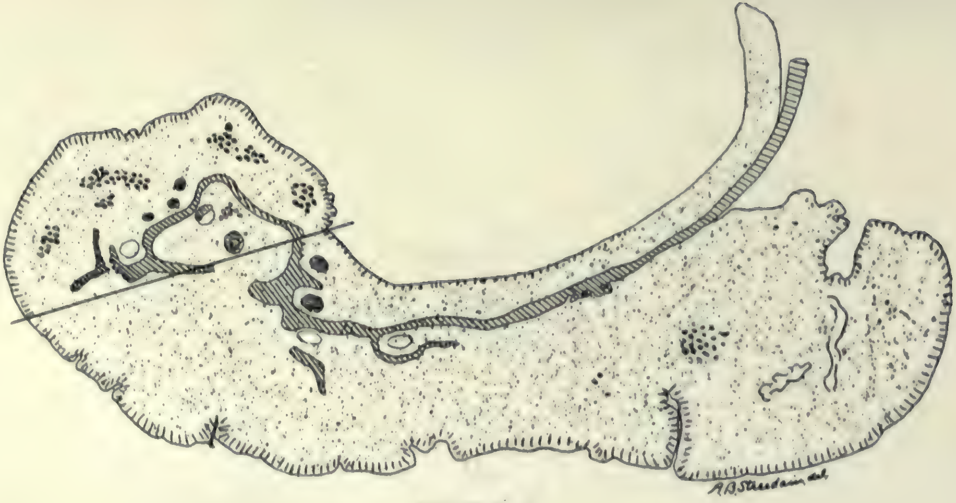


FIGURE 11.

Key to Figure 10.

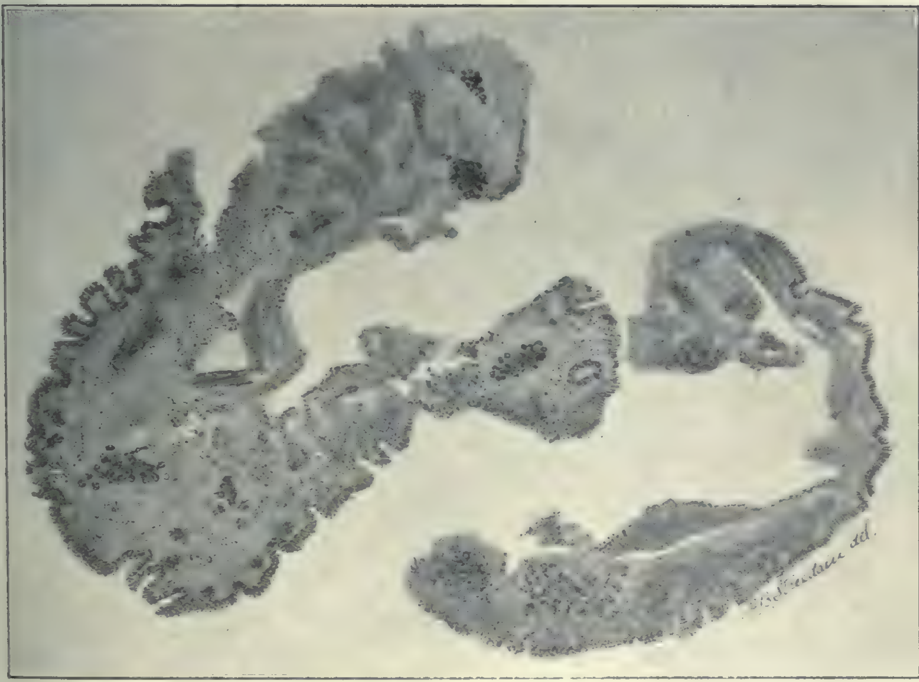


FIGURE 12.

Ethmoid cells. Chronic suppurative sinusitis. (Asthma.)

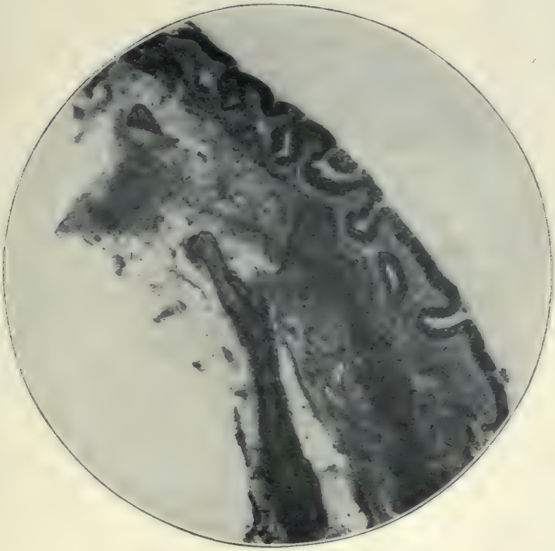


FIGURE 12A.

Necrosing ethmoiditis and chronic infiltrated hypertrophy of the mucous membrane.

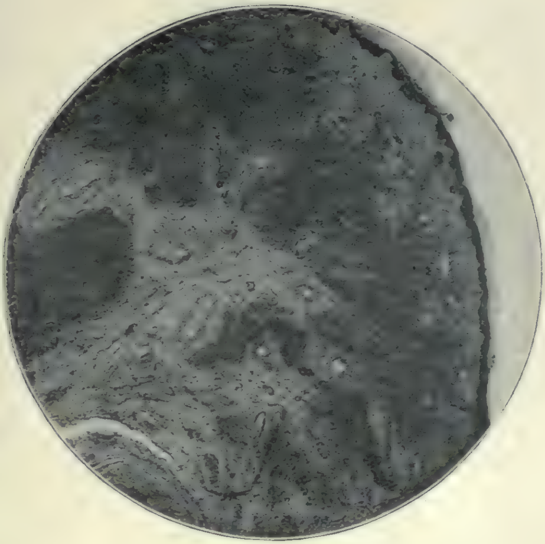


FIGURE 12B.

Chronic glandular hypertrophy of middle turbinate in chronic suppurative pansinusitis.

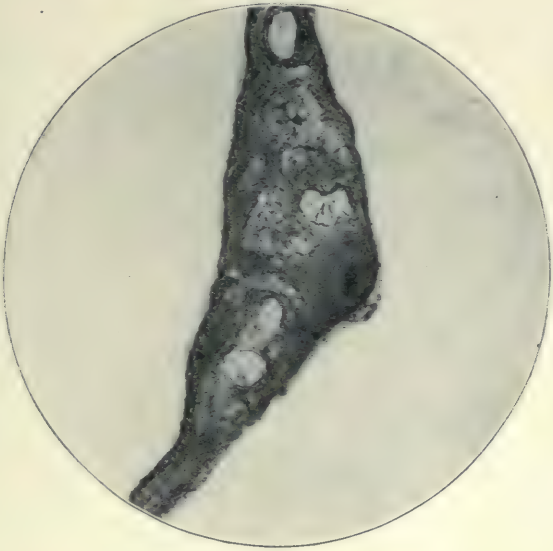


FIGURE 12C.

Rarifying ostitis of the ethmoid cell in nonsuppurative sinusitis.

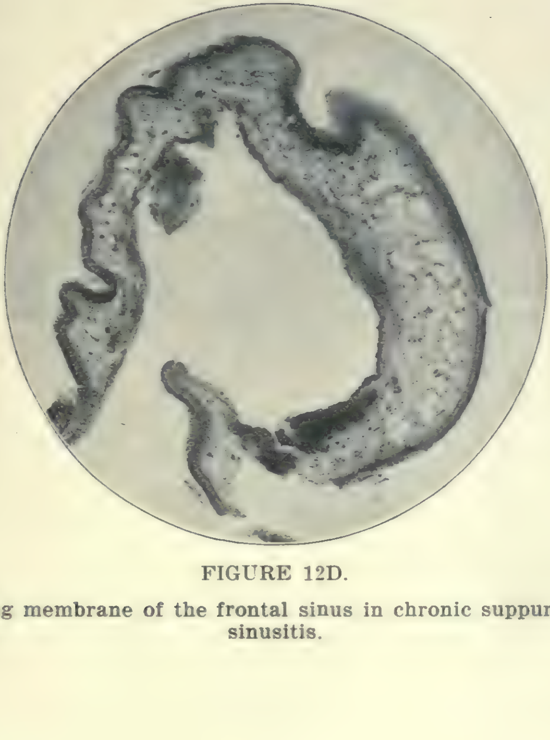


FIGURE 12D.

Lining membrane of the frontal sinus in chronic suppurative sinusitis.

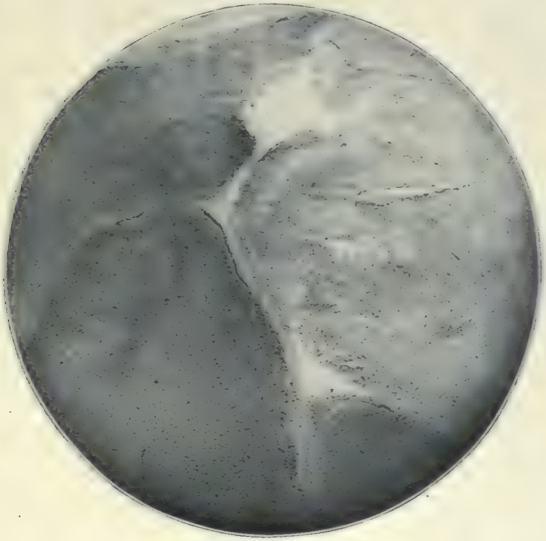


FIGURE 12E.

Pyogenic membrane lining the antrum of Highmore in chronic suppuration of all the sinuses.

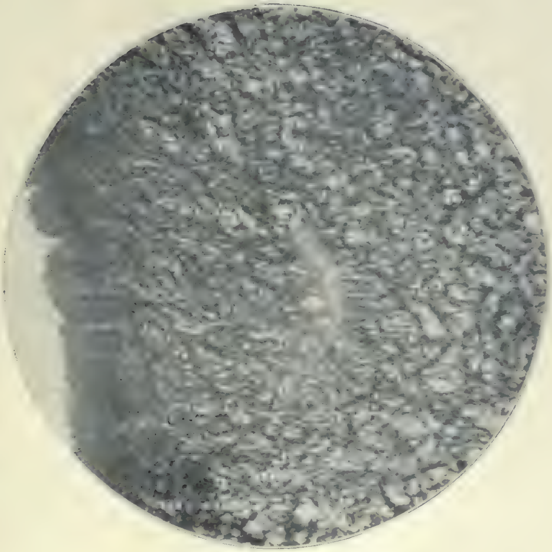


FIGURE 12F.

Solitary fibromyxomatous polypus of the frontal sinus in chronic suppurative pansinusitis.

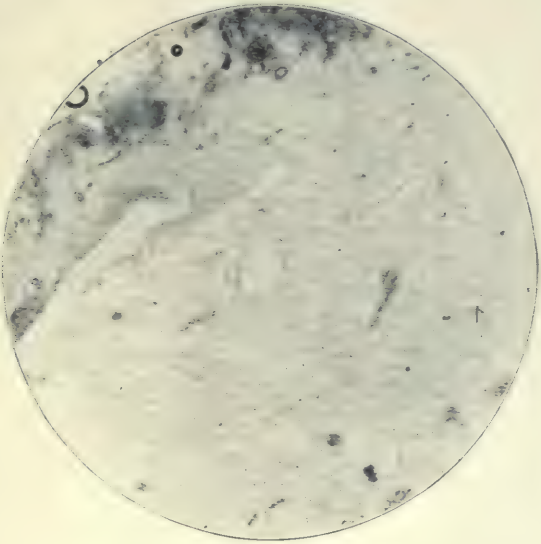


FIGURE 12G.

Inflammatory edema of a nasal polyp with cystic formation in chronic nonsuppurative sinusitis.

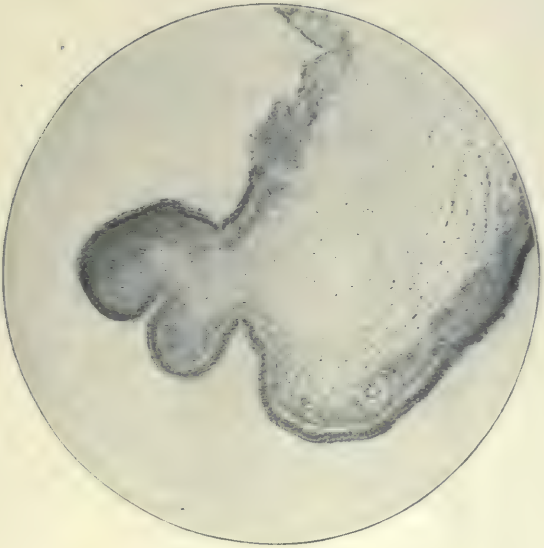


FIGURE 12H.

Inflammatory edema and hypertrophy of epithelium of a large nasal polypus in nonsuppurative sinusitis.

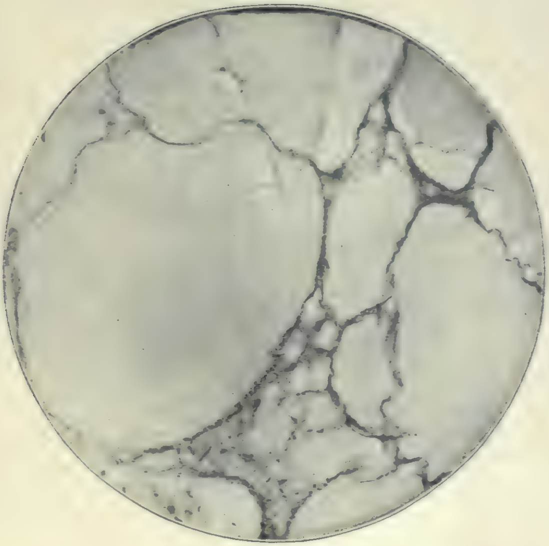


FIGURE 121.

Cystic formation in a nasal polyp in a nonsuppurative sinusitis.

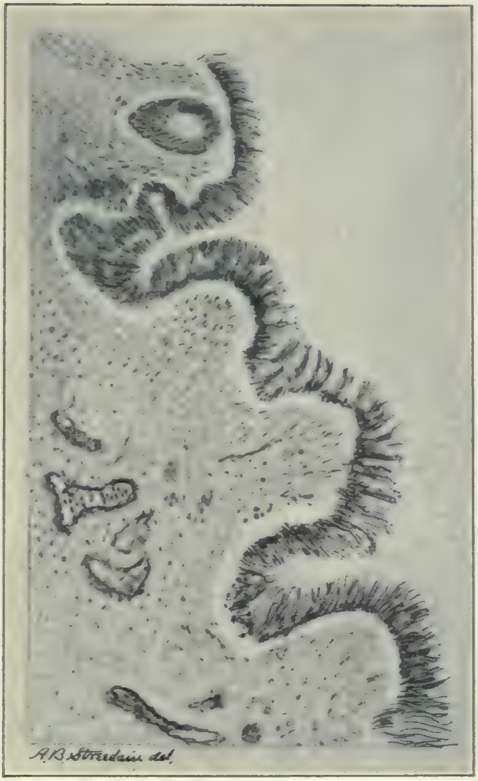


FIGURE 13.

Middle turbinate. Chronic nonsuppurative sinusitis. (Asthma.)

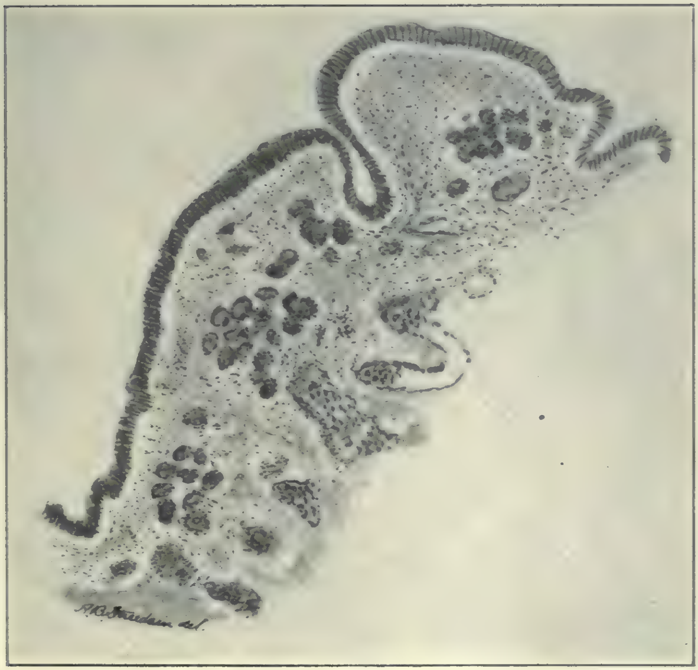


FIGURE 14.

Middle turbinate. Chronic nonsuppurative sinusitis. (Asthma.)

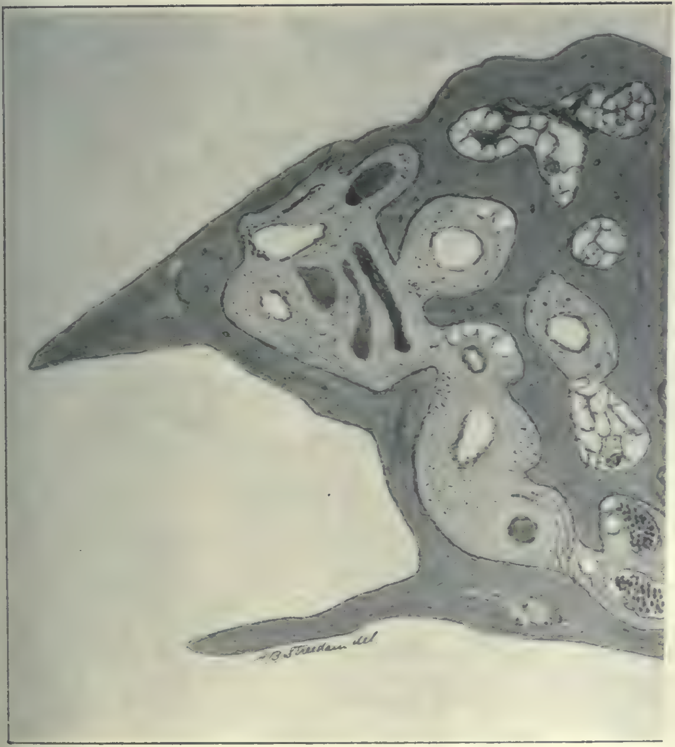


FIGURE 15.

Middle turbinate. Chronic nonsuppurative.

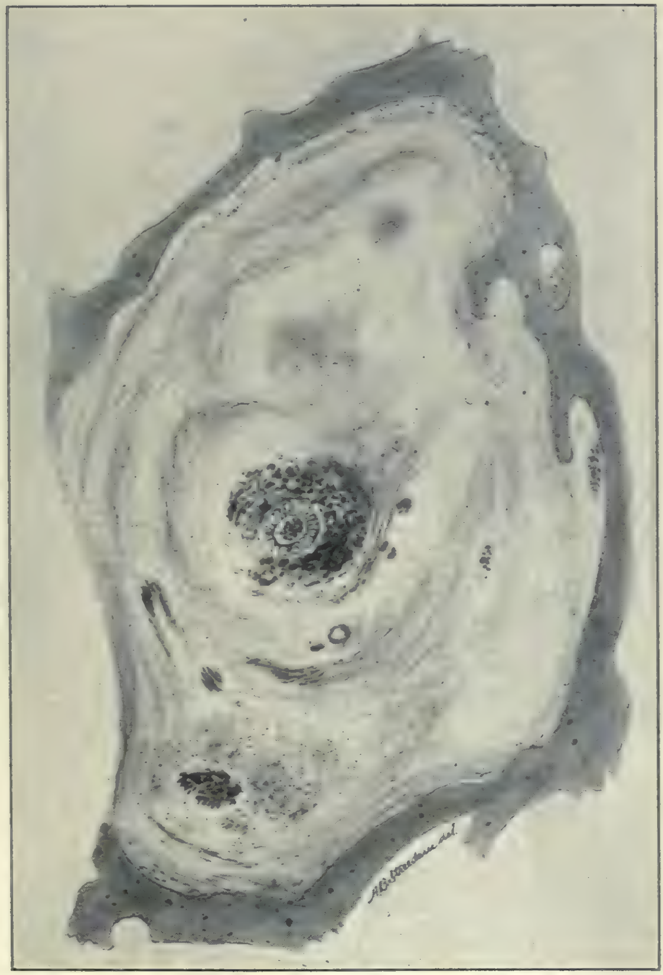


FIGURE 16.

Middle turbinate. Chronic nonsuppurative sinusitis. (Asthma.)

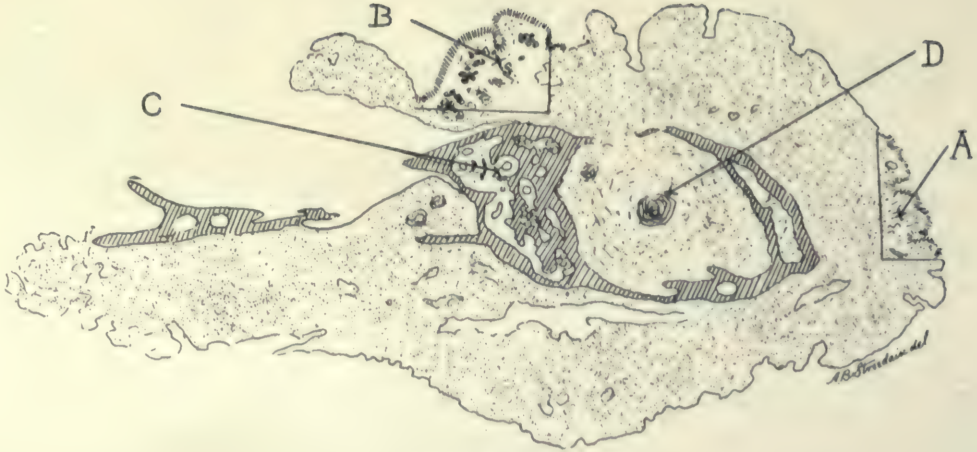


FIGURE 17.

Key to Figures 13; 14, 15 and 16.

LVII.

THE ENDONASAL ROUTE OF ATTACK IN HYPOPHYSEAL TUMOR CASES.

By W. E. Sauer, M. D.,

St. Louis.

As late as 1882 Hyrtl stated that the sphenoid cavities were entirely beyond the reach of hand or instrument. Today the modern rhinologist deals with these sinuses as a part of his regular routine, and even employs them as a route for entering the cranial cavity. After experimental research and clinical observation had proven that the disorders of the hypophysis were amenable to surgical intervention, the rhinologist was not long in proving that these diseases were within his province, in fact he has been able to show a lower mortality rate than others not working along this line.

The hypophysis has been reached by two different routes: the intracranial and the extracranial or transphenoidal. The intracranial route was first suggested by Canton and Paul, and later employed by Horsley. McArthur attempted to attack the hypophysis through the anterior cranial fossa in 1908, but failed. Bogoisolensky performed the first successful operation by this route in 1912. Schloffer, however, performed the first successful operation in 1907, employing the transphenoidal route. He found it necessary to remove practically the entire nasal interior, as well as temporarily to resect the external nose. This operation was later modified by Koenig, Knavel and Halsted, but to Hirsch of Vienna belongs the credit of developing the endonasal operation. He conceived the idea that the sella turcica could be opened through the nose without the preliminary operation of severing the external nose from its attachment and turning it to one side, as was done by Schloffer. The real point in Schloffer's operation was the opening of the sella turcica through the sphenoid. To accomplish this he and others found it necessary, in order to have sufficient room to work, to resect first and then to push aside the external nose. The real difficulty, as stated by both Schloffer and von Eiselberg, was to keep their bearings after they entered the ethmoid. It became extremely difficult for them to tell if they were still within the ethmoid or had reached the sphenoid. Von Eiselberg attempted to take his measurements from a radiograph of the patient. In one instance at least it was necessary to call on the anatomist Tandler to assist him in finding the sphenoid cavity.

Hirsch carried on some experiments on the cadaver, and attempted to open the sella without disturbing the nasal structures other than removing the middle turbinate. He succeeded in making an opening in the floor of the sella 1 cm. by 5 mm. wide, without injuring any of the surrounding structures. These experiments were later controlled by Dr. Erdheim of the pathologic institute of Vienna. Hirsch had also observed in all of his work on the sphenoid sinuses on the living, how much of the upper wall of the sinus could be seen and how relatively easy it would be, especially when there was a large sinus, to open the wall and if necessary to remove a part of it. His studies of a large number of radiographs of the skull still further confirmed his views.

Hirsch developed two methods of attack through the nose:

First, the ethmoidal route. This consists of the removal of the middle turbinate with the exenteration of the ethmoid labyrinth on the same side. This gives a free exposure of the anterior wall of the sphenoid. Its anterior wall is then removed as far as possible, and the upper wall or floor of the sella turcica is opened with a chisel, and this opening is enlarged with a suitable forceps.

The second is the septal route. This consists of a complete submucous resection in the usual way. In addition, the mucous membrane covering the anterior wall of the sphenoid is pushed to each side until the openings of the sinuses are visible. The entire bony septum having been removed, the openings of the sphenoid are enlarged and the partition between the two cavities is then removed. This gives a free access to the upper wall of the sphenoid, which is opened, and the opening enlarged as before. In this operation the work is done entirely between the two mucous membranes of the septum. If the middle turbinate must be removed, it should be done several weeks before. The advantage claimed for this route is that all the manipulations are carried on within a sterile cavity without entering the cavity of the nose. This, however, is not alwaays possible, because the mucous membrane is not so easily deflected from the anterior walls of the sphenoid, and as the mucous membrane is rather thin at this point, it is easily torn and the cavity of the nose is entered; at least this has occurred in my case.

About the same time that Hirsch was doing his work, West of Baltimore brought forth a method which he worked out on the cadaver. His method consists of a partial removal of both inferior and middle turbinates. Enough of the turbinate is removed to give a good view of the anterior wall of the sphenoid. Without elevating the mucous membrane of the septum, he makes a vertical incision through the cartilaginous septum, and then by means of a chisel makes a cut, beginning at the upper end of the first incision and extending to the upper part of the posterior end of the septum. A second is made from one-half to three-fourths inch below and parallel to the upper cut. The bone is then removed; this gives a free access to the sphenoid and gives a large space to work in. The anterior wall of the sphenoid is then removed with a chisel. The septum of the sphenoid is next removed and the sella opened, as in the Hirsch operation.

Cushing makes an incision under the lip, and then does a complete resection of the septum through this incision. He claims that in starting under the lip there is about twice as much room than that secured by the Hirsch method. Cushing calls attention to the difficulty in avoiding a tear in the mucous membrane in elevating it from the cartilaginous septum. He also points out the possibility of entering the ethmoid and mistaking it for the sphenoid. In one case the cribriform plate was opened, owing to the fact that the angle of attack was directed too high. This error is not likely to occur with a patient under cocain anesthesia, if the Hirsch or West technic is followed.

The chief objection to the intranasal route has been the danger of infection, resulting in meningitis. It has been argued that the nose is not a sterile cavity, and that it is impossible to make it sterile. This, however, has not been borne out by experience. Hirsch has reported twenty-six cases of hypohyseal tumor which he has operated upon, with only three deaths. One death was due to a pneumonia; in another, a hemorrhage occurred into the third ventricle; and in the third, a meningitis. In this case the patient was a dement, who removed the packing himself. The results of Hirsch's operations are better than any series of operations reported by any other writer. Cushing had four deaths in twenty-nine cases. Von Eiselberg had four deaths out of fourteen cases. In thirty-two operations according to Knavel's method, six deaths occurred.

Spiess and Holmgren each report a case operated upon by the Hirsch method, with good results. Adding my own case to this makes a total of twenty-nine cases, with a mortality of 10.3 per cent as compared to Cushing's 13.7 per cent. Furthermore, Hirsch operated on one of his cases six times and another four times. These secondary operations should be considered in making up the percentage, as these operations exposed the patient to the same danger as the primary operations; when these are added it brings the mortality rate down to 8.1 per cent. These figures would prove that the danger of infection is more apparent than real. Schloffer has further shown that the hypophysis differs from other portions of the brain, in that while all the other portions of the brain are covered by the pia, arachnoid and dura, the sella portion of the hypophysis is covered only by the dura. The subarachnoid space is not opened when only the sella portion of the tumor is removed, thereby greatly lessening the danger of infection.

Cushing states that as a matter of fact the risk of inducing an infectious meningitis is comparatively slight when the turbinates are not removed and the mucous membrane is not lacerated. Even the existing risk is greatly lessened by the application of Crow's discovery, that hexamethylenamin finds its way into the cerebrospinal spaces. This drug is given in doses up to sixty grains in twenty-four hours, by mouth, on the day preceding and for some days after the operation. Von Eiselberg reports two deaths from an infectious meningitis following his modification of the Schloffer operation. He attributes this infection to an existing acute rhinitis which had been overlooked or underestimated by him. One case died within thirty hours and the other four days after the operation.

In looking over the literature I find reports of thirty cases operated upon by the endonasal route; twenty-six of these were performed by Hirsch of Vienna, one by Spiess, one by Holmgren, one by Pierce, and one by Morshik. In summing up his results Hirsch states that three deaths occurred: one from pneumonia, one from hemorrhage into the third ventricle, and one from meningitis. In three cases there was no improvement following the operation; in two of these cases there was a marked defect in vision of long standing; the other case was an old case of acromegaly. He concluded that in these cases the principal parts of the tumor had grown upward, and the small parts removed had no influence on the symptoms, or else the optic nerves had been destroyed. In five of the cases the relief obtained was only of short duration—two to six months. In sixteen cases the results were good and the improvement obtained has been maintained. In this group are a number of cases in which there has been a marked improvement in vision as well as improvement in the general symptoms. In one case two years have elapsed since the operation with no recurrence of the symptoms.

Pierce of Chicago reports a case of increasing blindness. diplopia and headache, in which a growth in the sphenoid sinuses was suspected. The middle turbinate was removed and the ethmoid cells were opened. The anterior wall of the sphenoid wall was then removed and a slight amount of mucopus was evacuated. The lining membrane of the cavity was pale and smooth. The striking feature was the small anteroposterior diameter of the cavity. When touched with a probe it conveyed a sensation of bogginess; marked improvement followed this decompression. The case was later operated on by Dr. Halsted, by a modified Schloffer.

Spiess reports a case in a patient twenty-eight years of age, in which the diagnosis had been confirmed by the X-ray. He followed the technic of Hirsch. and found, instead of the posterior wall of the sphenoid cavity, a tumor about the size of a hazel nut. which proved to be a cyst. In attempting to remove this cyst it was ruptured. The contents were removed and as much of the wall as possible was curetted away. The patient ran a temperature for a time, but made a good recovery. Vision was restored, with marked improvement in the general symptoms.

Holmgren reports a case of a man aged forty, who complained of headache with marked impairment of vision. He had a bitemporal hemianopsia, contracted visual field. X-ray revealed a destruction of the bone beneath the sella turcica. Both middle turbinates were removed. A submucous resection of the septum was done. The anterior wall of the sphenoid was removed. The tumor had destroyed a part of the posterior wall of the sphenoid. No attempt was made to remove the tumor. Marked improvement in vision and general symptoms followed.

The case operated upon by Morshik was a patient of von Eiselberg's, in which the latter had done a temporary resection of the nose; he opened the ethmoid and a quantity of mucopus was found. A pulsating tumor was seen in the region of the sphenoid, but owing to the unexpected presence of pus in the ethmoid, von Eiselberg decided not to attack the tumor; the wound was packed with gauze, the nose was stitched back in place and the gauze tampon removed on the seventh day. Dr. Morshik was called in to see the case; he found large quantities of pus in the epipharynx. Four weeks later the patient developed an acute angina, which necessitated the postponement of the operation. The mucopurulent discharge finally ceased, and two months after the first operation Morshik attempted the endonasal removal of the tumor. The patient was given a morphin scopolamin injection one hour before the operation, and the mucous membrane infiltrated with a 1 per cent novocain solution. This tumor was found to be covered with granulations which had to be removed. The opening in the sphenoid was enlarged. On attempting to remove the tumor it fell to pieces on the floor of the nose. A free hemorrhage followed; this was controlled by packing with gauze; the gauze was removed after a few moments and a probe introduced. The probe passed into the cranium for quite a distance without any resistance. The gauze was again gently packed into the wound and removed one hour later. The tumor proved to be a sarcoma. The evening after the operation the patient had a high temperature with headache. Patient died in forty-eight hours, from meningitis.

HYPOPHYSEAL TUMOR WITH ACROMEGALY.

In conclusion I wish to report the following case referred by Dr. Behrens, to whom I am also indebted for the history and the physical findings:

S. D., aged thirty-two years, white; coal miner formerly; saloon keeper now. Lithuanian (Russian Poland).

Complaint.—Was sent to me in October, 1911, because of palpitation of and oppression over heart, causing anxiety and fear; weak generally; growing weaker. Also had gastrointestinal disturbances without undue provocation.

Family history.—Father died at sixty-seven years of age, cause unknown; was of normal proportions. Mother living, age seventy-five years, and well; also of normal physique. Brother living and well, age forty-two years; large, over average height and weight. Sister died at fifty-two years, cause unknown. Family were all large, but not ill proportioned. Knows of no nervous, cancerous, tuberculosis or kidney diseases in family.

Previous history.—General health splendid until seventeenth year of age. He has had the milder exanthemata of childhood, but no scarlet fever, diphtheria, rheumatism, chorea, typhoid fever, pneumonia or malaria. He denies venereal disease or sexual overindulgence, and has been moderate in habits as to tobacco or drink.

Personal history.—At the age of seventeen he began to have slight headaches, never severe, but at times periodical and constant, frontal in character and especially early mornings. Some ten years ago the symptoms increased, with visual disturbances added, most prominent in left eye; then gradually the right eye began to show similar disturbances, but he cannot see as well now with the left eye. He suffers from cardiac excitability.

Examination.—Head—No deafness, no ear discharge, odd feelings in head.

Cardiorespiratory.—No cough, no dyspnea, no edema; perspires easily.

Gastrointestinal.—Good appetite, some dull pains in stomach and around heart, no nausea, no vomiting, eructation at times of sour material; bowels regular.

Genitourinary.—Urinates regularly, no gonorrhea or syphilis.

Neuromuscular.—No convulsions, numbness or peripheral pains; has normal sexual power and desires. Quit work as a miner because of weakness and cardiac distress.

Examination.—Patient large, thick set, white male adult.

Abdomen.—Negative; no palpable enlargements of spleen or liver. Penis shows no scars, of good development; testes likewise of normal consistency. Normal knee jerk, no Babinsky, no clonus, no edema of legs or ankles; some veins enlarged. Both feet large, large toes, quite abnormally thick, others above normal, bones of legs and thighs not irregular or equally or portionately enlarged. Spade-like hands, large fingers, short thick thumb 9 cm., middle finger 9 cm. Teeth show marked separation owing to enlargement of lower jaw. Tongue is also large.

Ophthalmic examination.—Pupils not dilated, respond to light, discs unusually bright and shining, optic atrophy slight, no vessel engorgement, not tortuous. Vision good, right eye 15/12, left 15/200.

During year of observation very little difference in any condition, mental or physical, was noted, with the exception of marked decrease in vision in left eye.

Nose.—No discharge.

Over skin of thorax, abdomen and back are numerous nodules, firm, fibrous-like in feel, movable, not tender.

The indication for operation was the increasing loss of vision in the left eye. The patient was sent to the Deaconess Hospital on November 28th, and on the following day, after the usual preparations for a submucous resection of the septum, he was sent to the operating room. The septum and the region of the anterior wall of the sphenoid were carefully cocainized with a 20 per cent solution of cocain. In addition, a one-half per cent solution of cocain was injected on each side of the septum near its posterior end. The septum was deviated to the left anteriorly, and the right posteriorly. The middle turbinate was removed on the left side, after which a submucous resection was done in the usual way. The mucous membrane on the anterior wall of the sphenoid was pushed to the side until the openings of the sphenoid were reached. These openings were enlarged and the partition between the two sphenoid cavities was then removed. The upper wall of the sphenoid was found to be much lower than usual; it appeared to be bulging down into the cavity. This wall of the sphenoid cavity was then opened by means of a chisel, and the opening enlarged by means of a punch forceps. The bone was not as thin as I had expected to find it, but there was no difficulty in removing it. An opening about 1 cm. by ¾ cm. was made. The dura was incised by means of a sickle-shaped knife. This incision was very painful, but I managed to make a crucial incision; the patient requested that I discontinue.

The mucous membrane covering the anterior wall of the sphenoid was torn to such an extent that I packed the nose in the usual way, and did not pack between the mucous membrane, as recommended by Hirsch. The pack was removed at the end of twenty-four hours; there was no unusual bleeding or any other usual symptoms following the operation. The patient noticed a marked difference in the heart's action, and stated that he felt much lighter; he particularly noticed that the foul odor he had previously noticed had disappeared. The patient left the hospital ten days later.

An ocular examination by Dr. Parker shows the following: Conjunctiva, cornea, anterior chamber and iris, normal. Reaction to light better in right than left. Accommodation better in right than left. Visual acuity in right normal, left 17/120: could not be improved.

Ophthalmoscopic examination.—Right showed very slight beginning atrophy; left fundus on the temporal side looked clouded and markedly thinned, and in places disappearing entirely. Nasal half vessels much more easily defined.

Color field.—Much contracted, especially temporal side of left eye, also interlacing.

December 10, 1912.—Vision, right normal, while left was much improved; vision 17/60.

January 2, 1913.—Patient feeling and looking much brighter; right eye, vision normal; left 17/50.

February 1, 1913.—Right normal, left still improving; vision 17/40.

Eleven months have elapsed since the operation, and his vision in the right eye is normal, while the vision in the left eye remains at 17/40. The patient has been working as a day laborer, unloading ice, etc., without any discomfort, whereas before the operation he could scarcely walk a block without having to stop to rest. He now wears a smaller hat as well as smaller shoes. He is also able to bring his upper and lower incisors together, while before the operation the lower teeth protruded about a half inch. Should there be a recurrence of the symptoms, I will attempt to remove a part of the tumor.

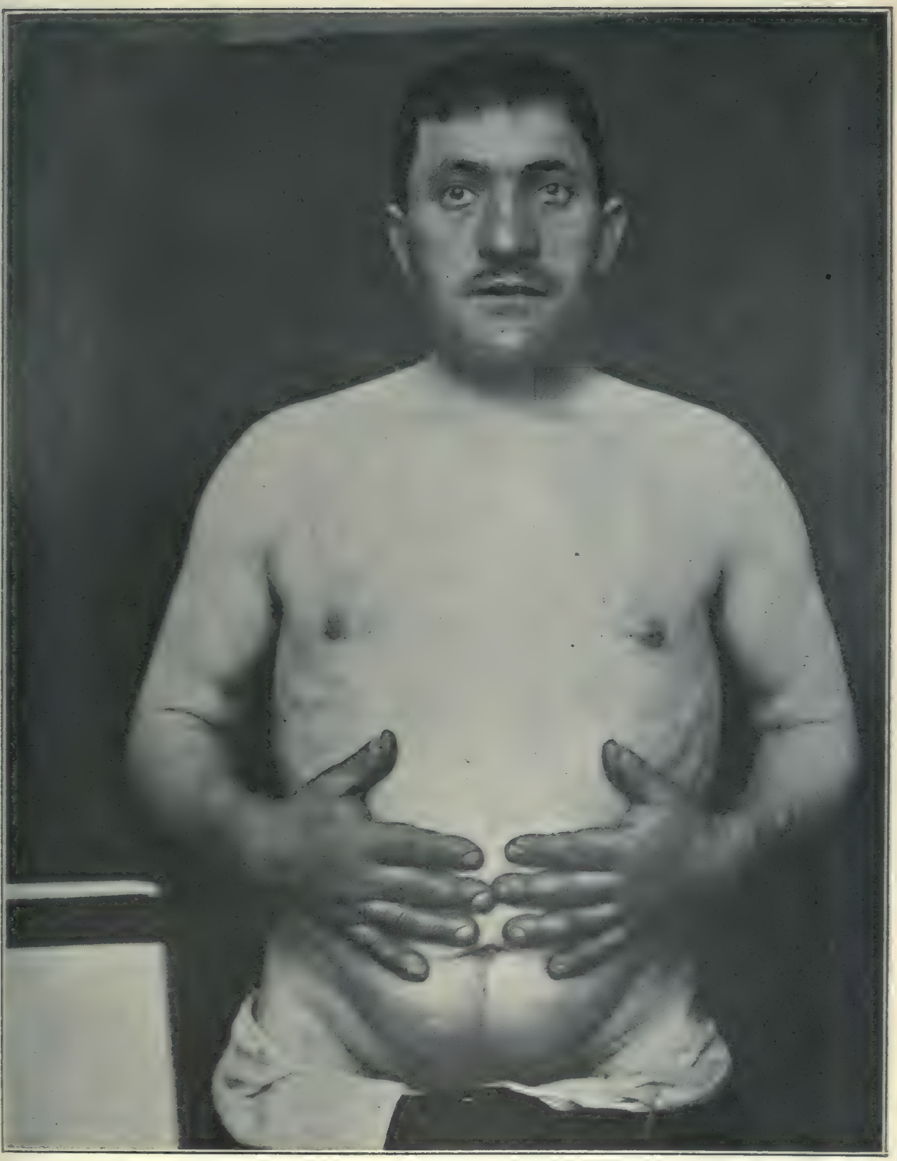


FIGURE 1.

Showing relative size of hands and body.

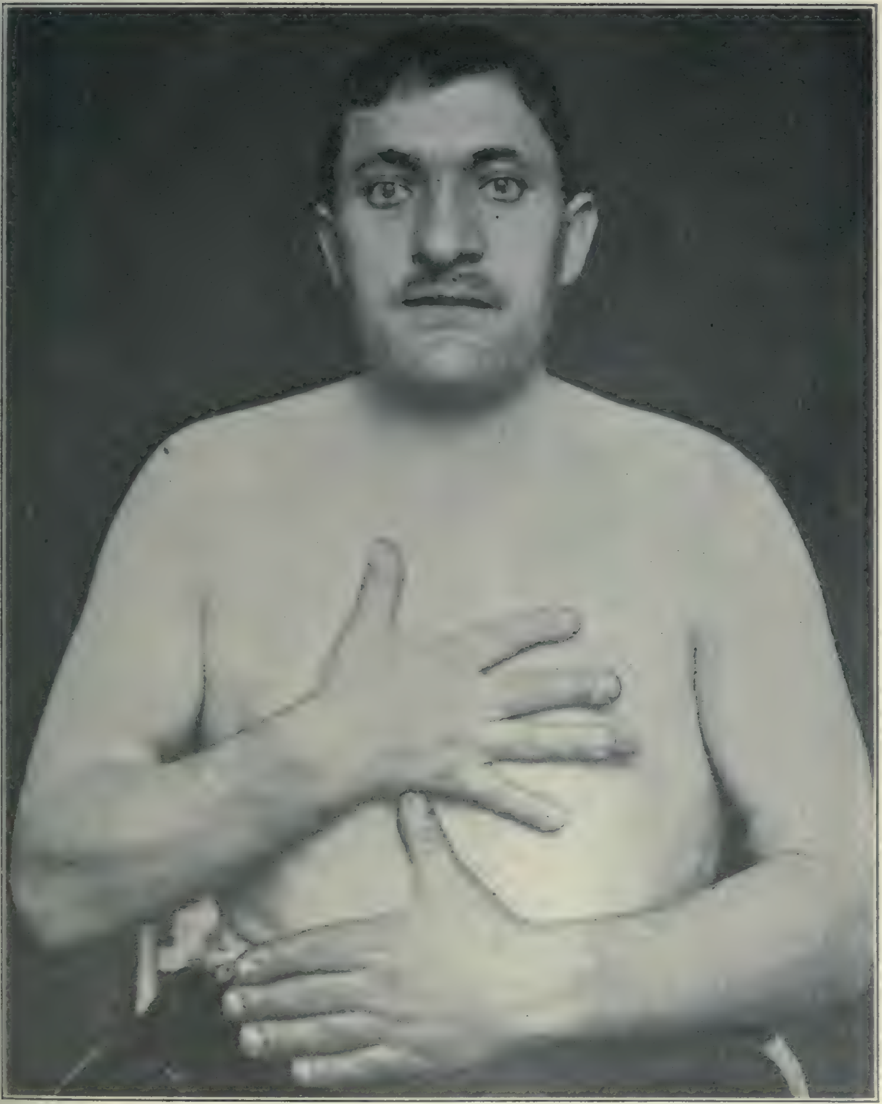


FIGURE 2.

Showing typical hands, chin and facial expression.

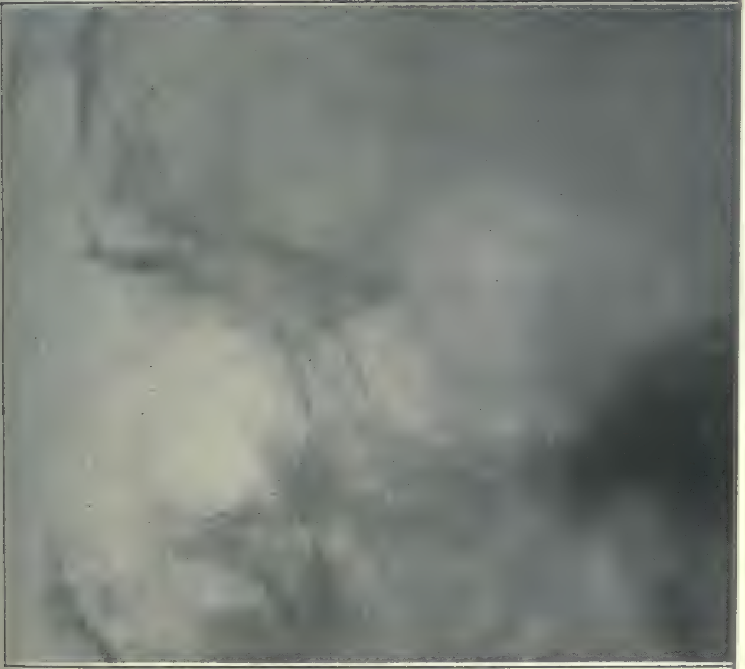


FIGURE 3.

Radiograph showing large sella turcica.

LVIII.

CAVERNOUS SINUS THROMBOSIS DUE TO ORGANISMS OF THE STREPTOTHRIX GROUP.

By Henry Horn, M. D.,

San Francisco.

Since 1907, when Löhlein reported his case of brain abscess resulting from a streptothrix infection, nothing of importance has appeared in the literature concerning this phase of the disease.

It is my good fortune to be able to add a little to the knowledge of this interesting subject and to report a case which seems to be absolutely unique in the scattered literature of the streptothrix infections.

Although there are numerous records of cases where the cause of death was proved to be an infection from streptothrix in various parts of the body, the cases combined with intracranial complications are comparatively rare, and, what interests us from the standpoint of otologists, intracranial complications originating from ear infections seem to be absolutely unknown.

In this article our interest has naturally been centered only on those cases in which the brain and its meninges have been involved.

The first case of this nature to be fully described and worked out bacteriologically was reported by Eppinger in 1890.[57] He was able to cultivate a mould from a metastatic abscess in the brain, and named the organism cladothrix asteroides, but this has since been identified as a true streptothrix and is now called streptothrix Eppengeri. He found this organism pathogenic for rabbits and guinea pigs, and was also the first to see the very great resemblance to tubercular processes and therefore designated the disease as pseudotuberculosis cladothricia, a similarity which Flexner studied later and agreed to. In fact it is through this research that the recent work on the true botanical place of the tubercle bacillus was started.

In the same year Armquist also isolated a streptothrix from a meningeal exudate which occurred in connection with other mycelial forms.

Various fatal cases of streptothrix infections were now reported. Tschierschke (1891), Hesse (1892), Carten, Gruber (1893), and finally Riviere and Sabarez, in 1894, reported two cases, one of which again showed a metastatic brain abscess originating in a probable lung abscess. The streptothrix was isolated from both abscesses. In the same year Ferre and Faquet also isolated the organism from a brain abscess. Many interesting cases, not of intracranial origin, were reported by various authors, and again, in 1899, Ferrari described a metastatic brain abscess of this character, probably originating in broken down bronchial glands.

The next case which interests us was that of Chiari, who reported two cases of bronchiectasia which were complicated with meningitis. In 1903 both Horst and Löhlein reported a case in which multiple abscesses due to a streptothrix infection were found at autopsy, and again, in 1907, Löhlein had the good fortune to be able to report still another case of brain abscess of the same origin. From this time up to the present writing there seems to have been no other cases reported, and it can, therefore, be seen that the number of cases of brain complications following streptothrix infections is extremely small, probably not over ten all told; and as far as a careful review of the literature shows, none at all that started in the middle ear and later led to an involvement of the great blood sinuses of the brain.

The morphologic position of the streptothrix group is to some extent an unsettled problem, but it seems as if we ought to be willing to accept the carefully worked out life history, as given by Foulerton, who does not consider that the group should be classed as bacteria but as moulds.

If we look over the literature of the ear moulds, we find that the majority of the cases reported have usually been classed under the heading of mycosis, aspergillus or allied forms. These are clinically and morphologically unrelated to the streptothrix group, and as far as is known have never led to intracranial complications. They are generally confined to the external auditory canal, but in a case reported by Haug, he described the mould as extending into the cells of the mastoid process, and only a few cases of middle ear infection have been traced to this source.

Like many other botanical groups, the nomenclature in the earlier works on this subject has been very loosely applied, and, therefore, there is considerable confusion as to whether the streptothrix is related to the actinimycoses. This, however, is not the case, for in the life history of the streptothrix there are such marked differences that their identity with the latter group is at once excluded.

As nearly as can be determined, the name streptothrix was first utilized by Corda in 1834, but the identify of his organism is somewhat in doubt. Probably the mould isolated by Cohn (1874) from the lacrimal canal is the same as we today recognize under the title of streptothrix. The further historical development of the subject has been worked out in Foulerton's article and need not be repeated here.

Inasmuch as it is the opinion of the author that this organism is far more common in otologic infections than has generally been reported, a short description of the morphologic characteristics will not be out of place. The description is borrowed freely from Foulerton's article and is here added, as it would seem practical to have the first article dealing with the subject from the otologic standpoint as complete as possible, and thereby obviate the necessity of reference to the original sources.

Starting from the original spherical spore, growing in artificial media, under suitable conditions of warmth and moisture, two or three small buds project and, gradually elongating, become the primary filaments of the mycelial mass. From the primary filaments lateral branches bud out, and from these secondary branches other lateral filaments again grow out by a repetition of the budding process, and in this manner a more or less dense and tangled tuft of wavy branching mycelium is formed.

The termination of the filaments presents various appearances in different species, and the appearance may not be constant in the same species; the end of the filaments may be pointed or rounded off, or may present small spherical or Indian-club shaped enlargements. The adult mycelium presents a homogeneous appearance when stained with the ordinary basic anilin dyes or by Gram's method; when segmentation occurs there is an alteration of the staining properties, resulting from a degeneration of shorter or longer segments of the constituent filaments occurring at irregular intervals along their length. The degenerated portions of the filaments no longer retain the stain when treated by Gram's method, and stain but faintly with basic anilin dyes employed in the usual way. Fragmentation of the mycelium follows on segmentation, the degenerated segments gradually disappear, and in the place of the original tuft of branching mycelium there are collections of separate, shorter or longer, lengths of the filaments, which may appear as straight or slightly curved rods, or may retain the wavy outline of the original mycelium, or may present a well-marked corkscrew or spirillum shape.

It is quite certain that the rod segments never give rise directly to new rod segments by a process which is in any way analogous to the fission process which is a characteristic of the bacillary forms of the schizomycetes.

From the above description and from observation it can be stated that the morphology of the streptothrix is widely different at different stages.

In some of the pathogenetic species of streptotricheæ it is impossible to trace the complete morphology on any sort of media, the mycelium develops very slowly, and segmentation and fragmentation of the filaments appear to occur coincidently with the growth. In such cases the ordinary cover glass preparation will show little more than closely packed masses of rod segments which often have a beaded appearance, the whole closely resembling what is seen in a similar preparation from a culture of bacillus diphtheriæ or bacillus tuberculosis.

With but a few dissenting voices, the place which the genus streptothrix belongs in botanical classification is among the hyphomycetes or mould fungi. The dissenters from this view would regard the branching mycelium, the rod segments, and the spherical spores as merely casual morphologic variations of a highly pleomorphic fission fungus, which under different conditions may assume a branching filamentous, a spirillar, a bacillary, or coccal form.

The streptotricheæ, both the saprophytic and the pathogenetic forms, are very widely distributed in nature. In the higher animals we have the streptothrix bovis communis and streptothrix nocardii, occurring commonly in cattle. Numerous other species have been described, but as yet no carefully worked out case of these species as occurring in man has been found.

One or two fairly dependable cases of streptothrix bovis communis have been reported as occurring in man, but other than these few cases which might point to the lower animals as a source of infection, we are at loss to give a satisfactory explanation of the original source of the infection.

The frequency of primary streptothrix infection of the mouth in cattle, resulting in the formation of small nodules situated immediately under, and sometimes actually invading, the mucous surface of the dorsum of the tongue, has obviously suggested that herbage may be a special habitat of the streptotricheæ and a possible source of infection. Further, the general belief that these infections are especially liable to occur among those whose occupations bring them into frequent contact with dust emanating from dried grass, is to some extent supported by a consideration of the habits and occupation of thirty-nine individuals above mentioned. Thus, there were four men employed in stables, constantly handling hay and straw, and possibly addicted to the habit, prevalent among those employed in stables, of chewing pieces of straw. Two of the patients were gardeners, two cases occurred in bakers, and one patient was a dairy maid.

There are certain new features covering the pathogenetic action of this organism in the case about to be described which make it necessary to refer once more to the general literature and to show what the generally accepted opinion is regarding this point.

The only cases which are at all similar to ours are the ones which refer to intracranial complications. Here the occurrence of the metastatic brain abscess is the predominating type. As seen in other parts of the body, the infection causes the development of nodules and larger masses of granulomatous tissue around the invading parasites. These masses may cicatrize or break down into abscesses.

Speaking generally, it may be said that the streptotricheæ which are the cause of human infection are not of themselves apt to produce any very marked constitutional symptoms or effects, either by the secretion of actively virulent toxins or by the production of like bodies by decomposition of the tissue in which they may be growing.

The uncomplicated action of the streptotricheæ, then, is not usually manifested by severe general symptoms, but rather in the physical results of the tissues actually infected. Generally it is only when the granulomatous tissue, having broken down, has become the seat of infection, or secondary infection by pyogenic cocci or other organism which secrete toxins having a marked general effect, that the wasting and more severe constitutional symptoms which occur in advanced cases of streptotrichosis are seen.

This general review, then, will serve to introduce the case in hand and serve as a basis for the consideration of some new points which it seems to illustrate.

C. Q., Italian, twenty-five years old. Admitted to the San Francisco Polyclinic and Postgraduate School's service at the San Francisco City and County Hospital, June 15, 1912. Died on same date. The patient was admitted with the general diagnosis of gonorrheal ophthalmia.

Past History.—Sensorium of patient entirely clear, all questions answered by aid of interpreter. Patient has been ill for about forty-five days. At that time he had an attack of right sided earache with a discharge of reddish, foul smelling pus. He gives history of repeated attacks of otorrhea since childhood, with periods of what he considered complete cessation of all symptoms.

About twenty-one days ago he had an attack of vomiting, dizziness, sweating and unsteadiness of gait. He had no chills, but he himself noted stiffness of neck. At this time he was admitted to a local hospital, where he was treated for meningitis with five injections of the original Schafer's serum without any apparent benefit. June 11th, a beginning exophthalmus was noted. The nose was examined for a suspected sinus infection, but unfortunately the ears were not examined. At this time the eye grounds were pronounced normal. The patient was discharged from the hospital the following day because of lack of funds. June 13th, patient had acute attack of dizziness, nausea, vomiting, severe disturbance of equilibrium, followed by severe chill.

General Examination.—Small, delicately built man, unable to speak English, sensorium absolutely clear. Temperature 102,° pulse 100, full and bounding. Neck absolutely rigid. High grade of double exophthalmus. Incomplete closure of eyelids. Lies flat on back, takes no notice of surroundings, unless spoken to, and continuously moans from pain in head. Locates intense headache behind eyes and over frontal lobes.

Physical Examination.—Chest.—Loud crepitant rales on both sides.

Heart.—Sounds fairly strong, rapid and no murmurs.

Reflexes.—Very slight knee jerk right, absent left. Abdominal reflex absent. Tendon reflex absent. Kernig's sign present, positive and marked. No Babinsky.

Urine.—Slight amount of albumin, otherwise normal.

Blood.—Reds 4,500,000. Whites 45,000. Polymorphonuclear 84 per cent.

Spinal Puncture.—Fluid under great pressure, turbid, many white cells. Repeated stains show no bacteria and no growth on several media.

Examination of Eyes. (Dr. Vard Hulen.)—Marked double exophthalmus, equal on both sides. Eyes divergent. Intense chemosis, especially in lower part of ocular conjunctiva. Extremely well marked swelling between eyes, extending up over the skin of forehead. The skin is hot, red in color and brawny to the touch. There is marked pressure sensitiveness at this point, but nowhere else.

Pupils normal in size and reaction. Vision, O. D., 12/200; O. S., 8/200. Cornea clear except little haziness of both lower margins. Atropin dilates pupils evenly and widely.

Ophthalmoscopic Findings.—Extensive neuroretinitis with marked retinal edema. No signs of choked disc.

Clinical Diagnosis.—Secondary orbital cellulitis. No fluctuation of tissue around eyes. No diagnosis of intracranial complications possible from background findings.

Examination of Ears. (Dr. Henry Horn.)—Left ear normal. Right ear: Very slight tenderness over right mastoid, no swelling or edema. Some tenderness along right jugular. Right submaxillary gland somewhat swollen. External canal filled with whitish débris, small amount of pus, no odor. Ear washed. Total destruction of drum; middle ear some smaller than usual on account of swollen mucous membrane. Upper part of external canal somewhat sunken. Weber to left. Bone conduction slightly shortened (?). Rinné negative. Hears loud shouts, with Bárány apparatus in other ear. Five minutes' irrigation with cold water produces no nystagmus and no dizziness. No spontaneous nystagmus.

Operation.—Ether narcosis, one hour and thirty minutes. Patient left table in fine shape. Pulse and respiration good. Rapid sinking and death after few hours.

The fact that the eye examination gave the impression that the exophthalmus was not due to an intracranial complication, but due to an orbital cellulitis, and the evident involvement of the sigmoid sinus and right jugular, the fair condition of the patient and the free sensorium, combined with the absolutely fatal prognosis without operative interference, were the indications for operation.

Mastoid Operation.—Usual incision. Soft parts not swollen. Few if any cells in mastoid tip involved. Sinus located far forward and only a few millimeters from the external bony canal. In spite of most careful search no antrum could be found at operation. (Its absence was afterwards confirmed at autopsy.) Middle ear filled with whitish débris, which did not give the impression of cholesteatoma, and on the other hand did not resemble any pus collection which the author had ever seen.

Sinus Operation.—Sinus laid free for an inch or more. The wall of the sigmoid sinus was entirely broken down, the clot had disintegrated and was filled with yellowish débris without odor. The thrombotic process evidently extended into the bulb and the same was rapidly and thoroughly laid free. It was seen that the jugular would have to be explored, so the uncovering of the lateral sinus was first extended about an inch toward the torcular, or until free bleeding occurred, and this was controlled with iodoform gauze tampon. The character of the thrombus changed completely as the torcular was approached, from a septic, markedly purulent mass to a reddish blood clot, which was not broken down. The free bleeding then occurred.

Ligation of the Jugular.—Incision from angle of jaw to clavicle, along the anterior border of the sternomastoid muscle. Large mass of infected glands in upper third of neck had to be dissected free in order to expose jugular. Facial vein ligated, although it was not thrombosed. Ligation as close to clavicle as possible, evidently well beyond thrombus. The entire vein was thrombosed with the same reddish clot as found near torcular, and was removed en bloc. Bulb washed out from above. Upper end of vein sewed to upper angle of skin incision, after the method of Alexander. Wound closed, free drainage from lower angle.

The operative findings proved without doubt that we had to do with a far more extensive sinus involvement than was possible to correct surgically, and as the patient's general condition was good he was sent to ward. Surgical shock showed itself almost immediately, and the patient died in a few hours without recovering consciousness.

The autopsy was made through the courtesy of Prof. Wm. Ophuls, of Stanford University, the findings were dictated to his own clerk, and the subsequent microscopic work was done by him personally. I take this opportunity of expressing my great gratitude for his generosity in allowing me to use the results of his work. There was no indication at the autopsy that we had to do with anything more than an ordinary septic infection, and it was only by the thorough method which all cases are carefully worked out in the pathologic laboratories of Stanford University that the true etiologic factor in this case was discovered.

Necropsy Report.—(Prof. Wm. Ophuls). Strongly built, rather poorly nourished man, of about thirty years of age. There is a very marked exophthalmus so that the lids cannot close. The opening of the lids at present is about 1 cm. The conjunctivæ are extremely hyperemic and edematous. There are large hemorrhages around the cornea. The lids are also congested and edematous. There is marked passive congestion of the lips, of left ear, and of upper part of neck. No palpable enlargement of the superficial lymph glands. No edema. There is a slight yellow discoloration of the conjunctivæ and skin of neck and shoulders.

In back of ear there is a deep wound about 5 cm. in diameter, and extending from that posteriorly, an incision about 6 cm. long. Wound is packed with gauze. The wound extends through the bone to the dura mater, and the dura is exposed over a large distance. The transverse sinus is opened for a distance of 1 cm., sigmoid sinus exposed to bulb of jugular vein.

On the right side of the neck there is a longitudinal incision reaching from the angle of the jaw down to the sternoclavicular joint on the right side. The wound is drained with cigarette drain. At the bottom of the wound the large vessels are exposed. The jugular vein has been resected practically for the whole distance; the upper end is sutured into the wound. One of the submaxillary lymph glands is moderately enlarged, grayish red and moist.

Skull cap normal. One of the pacchionian granulations near the longitudinal fissure extends very far into the bone, the posterior part of the longitudinal sinus has some soft clots and turbid bloody liquid. The dura mater is rather tense on both sides. Pia mater on right side shows marked diffuse hyperemia and some edema. The hyperemia is even more marked of left side. Pia mater at base is also hyperemic and edematous. The right anterior half of the pons shows a very distinct yellowish purulent infiltration; the same posteriorly, about the middle of lower surface of left hemisphere of cerebellum near the median line. On pressure upon cavernous sinus on either side there is a drop of pus discharged at its junction with the petrosus superior. The transverse sinus on both sides is also filled with thick purulent material. Same condition in the sigmoid sinus on the left side.

Soft tissues underneath the roof of the left orbit are full of pus, and there are numerous accumulations of pus underneath in the muscles. Same condition on the right side. Jugular vein on left side of neck is free. Right jugular vein is free below point of resection.

Brain normal, except for hyperemia and edema.

There is a marked atrophy of subcutaneous fatty tissue, the muscles are poorly developed. Stomach and liver in normal position. Transverse colon forms a V-shaped loop and very nearly reaches the symphysis. Few drops of fluid in peritoneal cavity.

Diaphragm.—Fifth rib both sides. Chest is rather poorly arched, long, symmetric. Heart, about three-fourths normal size. Soft postmortem clot in pulmonary artery. The heart valves are normal. Aorta is small. Many small yellow spots in intima. The bronchi are somewhat hyperemic and contain some mucus. The pulmonary tissue is normal. There is a small superficial scar at the right apex. Right lung same as the left.

Spleen measures one and one-half times normal size. Pulp is soft, of light grayish brown color.

Left adrenal is normal.

Left kidney slightly swollen. Cortex is somewhat opaque.

Right kidney, same as left.

Pancreas, duodenum, stomach, normal.

Liver, tissue hyperemic and moist, somewhat opaque.

The rest of the aorta like the beginning.

Diagnosis.—Purulent otitis media right side (streptothrix infection). Septic thrombosis of sigmoid sinus, of right jugular, of both transverse sinuses, posterior part of longitudinal sinus, both cavernous sinuses. Phlegmon of both orbits. Double exophthalmos. Purulent meningitis. Hypoplasia of heart and aorta. Fatty spots in intima. Parenchymatous degeneration of kidneys and liver. Septic spleen. Terminal icterus. Smears were made from infected sinuses, the meninges and from pus in left orbit. They all show many partly degenerated polymorphonuclear leucocytes, few lymphocytes, many large mononuclear phagocytes. In some of the smears, especially in those from the left orbit, a moderate number of very fine, Gram positive, somewhat irregularly staining filaments with true branching. They are single or matted together in branches, mostly extracellular. No other bacteria found, in spite of intense staining and careful search.

Anatomic Diagnosis.—Purulent otitis media on right side (streptothrix infection). Septic thrombosis of right sigmoid sinus, of the right jugular, of both transverse sinuses, posterior part of longitudinal sinus, left sigmoid sinus, both superior petrosal sinuses, both cavernous sinuses. Phlegmon of both orbits. Double exophthalmus. Purulent meningitis. Hypoplasia of heart, kidneys and liver. Septic spleen. Terminal icterus.

Epikrisis.—This "autopsia in vivo et in mortuo" would seem not only to enable us to add something to the scarce literature of the intracranial involvements following this somewhat rare infection due to the streptotricheæ, but also to draw a fairly clear picture of the method of involvement of the cavernous sinuses.

As far as a careful study of the literature goes, there has never been a case of streptothrix infection reported as having had its origin in the external ear.

The two most completely worked out cases are those of Eppinger and Löhlein. Both started as pulmonary conditions, and later by metastatic ways produced multiple brain abscesses. The infection of the great brain sinuses by this organism seems to be unknown.

The congenital absence of the antrum in our case is certainly very suggestive, and at the same time the extraordinary displacement forward of the sinus, to within a few millimeters of the external bony canal, leads one to the enticing theory that the infection of the right sigmoid sinus was by direct contiguity. This is borne out by the fact that one of the most characteristic pathologic features of a streptothrix infection is its socalled perforative tendency. If, for example, a small area of the pleura is involved, it may pass directly through the chest wall and give rise to an external abscess. With such a well known clinical tendency, we can well assume that the process started in the middle ear in early childhood, and by a slow process of ulceration broke down the few millimeters of protecting bone between it and the abnormally placed sinus, and then by the long contact with the wall of the sinus started the thrombotic process. The absence of the antrum, noted at the time of the operation and later confirmed at autopsy, would be additional proof that the process was always confined to the middle ear until later in life. The reason why the patient never received treatment for his condition was because the mastoid was never involved, and the sinus trouble was the first serious complication which he experiencd.

It is also to be noted that the fever curve was at all times remarkably low for a sinus complaint of such magnitude and apparent degree of purulent degeneration, and the very few attacks of chills would seem to indicate that a streptothrix infection of the sinuses does not give rise to such marked symptoms as does a streptococcus infection. Of course, the subsequent microscopic examination with the continued absence of true bacteria clearly explains why the very turbid spinal fluid gave no bacterial content. It was of course not examined for streptothrix at the time, as the condition was not then ever remotely suspected.

The condition of the cavernous sinus and the post- and periorbital tissues showed how impossible any satisfactory operative procedure would have been. The capsule of the orbit was so tense at the autopsy that when a puncture was made with a knife the pus spurted out under pressure. In this connection it is interesting to note how very slight were the changes observable with the ophthalmoscope.

It does not seem in place in this paper to discuss the various operative measures for attacking the infections of the cavernous sinuses. It would seem that the autopsy proves that in such a case as this operative procedure would have been worse than useless.

The autopsy findings, however, give us a certain right to theorize on the possible method by which the infection of these sinuses took place. In the diagrammatic figure shown in figure 1, the shaded parts shows the extent of the involvement. The fuller details were noted by me at the time of the autopsy for the specific purpose of tracing the mode of infection of the cavernous sinuses, and do not appear in the necropsy report of Prof. Ophuls.

The most striking thing about the appearance of the sinuses was the fact that two kinds of thrombi could be distinguished, which in all probability were different stages of the same process, but still distinct enough to show in which direction the thrombotic process was extending. First, there was the thick, yellow, semifluid purulent thrombus, which filled the right transverse, the right sigmoid, the right bulb and part of the upper part of the right jugular. About one inch from the torcular, or at the point that the operation was ended and free bleeding commenced, there was a reddish thrombus, that looked somewhat like a postmortem clot, but with greater consistency.

The purulent type extended up the right superior petrosal, did not seem to involve the inferior petrosal, invaded the sinus cavernosus and by way of the sinus circularis Ridleyi involved the whole region in a fluid purulent mass. From here the infection extended to the periorbital tissue, turning then into bags of pus under great pressure. The same purulent process now descended the left superior petrosal, involved the left sigmoid, where the purulent type was halted. The left transverse and the left bulbar region were again filled with the reddish, nonpurulent clot.

It seems fair to assume then that we have to deal here with a cavernous sinus involvement of originally otitic origin, the infection extending upwards along the right superior petrosal against the stream and then downward with the stream to the opposite side.

The careful bacteriologic examination of Prof. Ophuls would seem to indicate that the infection was a pure streptothrix infection, with the addition of very few if any real pus organisms. The preoperative bacteriologic examination would seem to substantiate this condition.

BIBLIOGRAPHY.*

52. Foulerton: The Pathology of Streptothrix Infections. Allbutt's System of Medicine, Vol. 2, Part 1, p. 302.

53. Horst: Zeitschr. f. Heilkunde, 1903, Vol. 24, p. 157.
(Full literature to date.)

54. Petruschky: Handbuch der pathogenen mikroorganismen. Edited by Kolle and Wassermann, 1903, Vol. 2, p. 832.

55. Engelhardt and Löhlein: Zur Kenntniss der Streptothrixpyämie. Deutsches Archiv. f. klin. Med., Vol. 75, 1903, p. 112.

56. Rosenbach: Ueber das Erysipeloid. Archiv. f. klin. Chirurgie, Vol. 36, 1887, p. 346.

57. Eppinger: Ueber eine neue pathogene Cladothrix und durch sie hervorgerufene Pseudotuberkulosis. Ziegler's Beiträge zur pathol. Anat., Vol. 9, 1891, p. 287.

58. Armquist: Zeitschr. f. Hygiene, Vol. 8.
59. Buchholtz: Zeitschr. f. Hygiene, Vol. 24.
60. Chiari: Zeitschr. f. Heilkunds, 1900.
Cohn: Biologie der Pflanzen, Vol. 1, Heft 3.
61. Cozollino: Zeitschr. f. Hygiene, Vol. 33.
62. Ferrari: Wiener klin. Wochenschr., 1899, No. 36.
63. Ferre and Faquet: Erg. d. allg. Path., Jahrgang 3, 1896.
64. Flexner: Centralblatt f. Bacteriologie, Vol. 24, p. 83.
65. Forster: Archiv. für Ophthalmologie, Vol. 15.
66. Fuchs: Vers. d. ophth. Gesellsch., 1896.
67. Garten: Zeitschr. f. Chirurgie, Vol. 41.
68. v. Graefe: Archiv. f. Ophth., Vol. 15.
69. Gruber: Archiv. f. Hygiene, 1873, Vol. 16.
70. Hesse: Deutsche Zeit. f. Chirurgie, Vol. 34.
71. Krause: Münchener med. Wochenschr., 1902, p. 749.
72. Löhlein: Münchener med. Wochenschr., 1902, p. 1161.
73. Naunyn: Mitteilungen aus der med. Klinik zu Königsberg.
74. Norris and Markin: Jour. of Experimental Med., Vol. 5, No. 2.
75. Riviere and Sabarez: Erg. d. allg. Path., Jahrgang 3, 1896.
76. Rosenbach: Archiv. f. klin. Chirurgie, Vol. 36.
77. Rullmann: Münchener med. Wochenschr., 1898, 1899, 1902.
78. Scheele and Petruschky: Congress f. innere Med., 1897.
79. Silberschmidt: Centralblatt f. Bacteriologie, 1900.
80. v. Schroeder: Klin. Monatblät. f. Augenheilkunde, 1894, 1896.

---

*The complete bibliography of this subject can be found in the above-mentioned article of Foulerton, up to 1906, and will not be repeated here. He gives 51 references, and the numbers in this article will continue from that number on, thereby giving a complete bibliography to date, April, 1913.

81. Tschierschke: Inaugural-Dissertation, Breslau, 1891.

82. Lubarsch: Ztschr. f. Hyg., Vol. 31, p. 187.

83. Schabad: Ztschr. f. Hyg., Vol. 47, p. 41.

84. Löhlein: Ueber Gehirnabszess durch Streptothrix. Münch. med. Wochenschr., 1907, p. 1523.

85. v. Ritter: Ueber einen fall von durch eine Streptothrix bedingter Pleuritis ulcerosa mit metatatischer Gehirnabszess. Prager med. Wochenschr., 1900, p. 525.

86. Claypole: On the Classification of the Streptothrices. Journ. Exp. Med., Vol. XVII, No. 1, 1913.

87. zur Nedden: Klin. Monatblät. f. Augenheilkunde, 1907, p. 152.

88. Hoffman: Gangrene of the Outer Ear Due to Streptothrix. Ztschr. f. Ohrenhkde., Vol. 51, 1906.

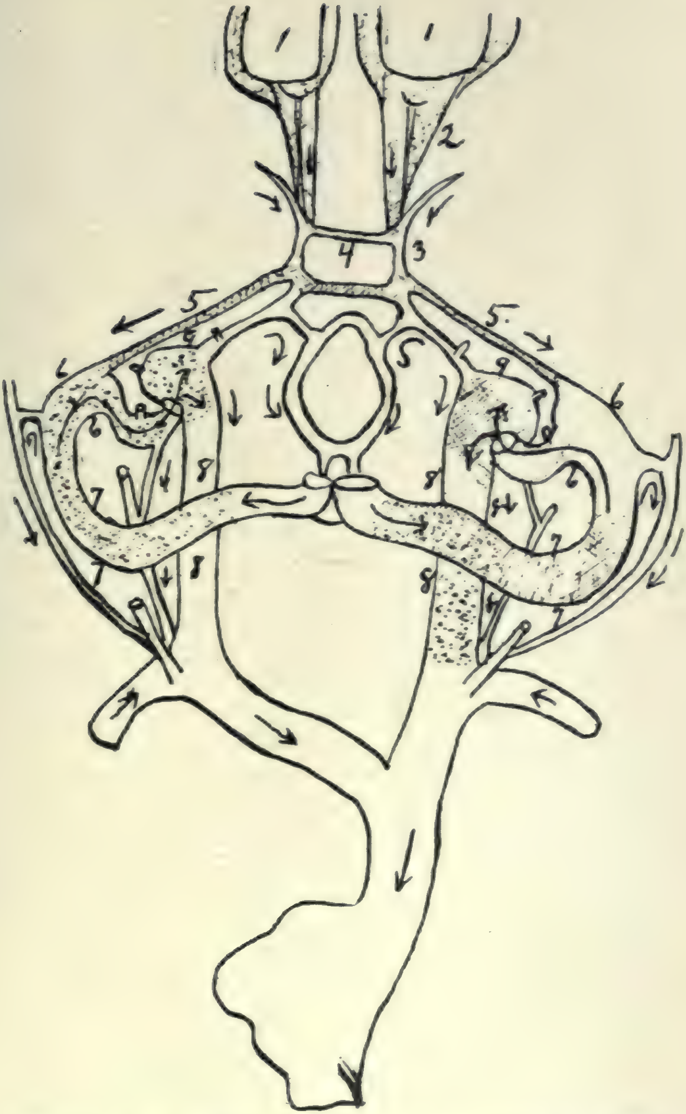


Diagram of the Sinuses. From Brühl's "Diseases of the Ear."

1. Bulbus oculi, with distended pus sac.
2. Vena ophthalmica, enormously distended.
3. Sinus cavernosus.
4. Sinus circularis Ridleyi.
5. Sinus petrosus superior.
6. Sinus sigmoideus.
7. Sinus lateralis.
8. Venæ jugularis.
9. Bulbus venæ jugularis.

LIX.

SUGGESTIVE POINTS OF ANALOGY BETWEEN OTOSCLEROSIS AND ARTHRITIS DEFORMANS.*

By John F. O'Malley, F. R. C. S.,

London.

The literature of chronic nonsuppurative diseases of the ear and joints is very extensive, and for the purpose of considering the analogy indicated by the title of this paper, I am putting forward the summary or gist of the views held by many of the best known authorities, and place them under headings which are chosen to emphasize the numerous points or features wherein these diseases seem to have a striking similarity.

It is now the custom for many writers to regard arthritis deformans as a group of diseases, namely, rheumatoid arthritis, osteoarthritis, and spondylitis deformans. As many of the most exhaustive descriptions of chronic nonsuppurative joint affections are given under the title arthritis deformans, I have retained this form of nomenclature.* It is, however, a little inconvenient, as it gives a combined picture of certain characteristics, such as the morbid changes, which some writers separate and place under the headings of rheumatoid and osteoarthritis respectively. Painter[1] calls the former atrophic and the latter hypertrophic arthritis.

The chronic nonsuppurative diseases of the ear which should be considered fully, to complete the analogy with arthritis deformans, are otosclerosis and chronic adhesive catarrhal processes, or, as Kerrison[2] calls the latter, chronic hyperplastic otitis media. As this would necessitate a much more extensive paper than the time at my disposal here permits, I have chosen otosclerosis alone to consider fully, but will refer in my conclusions to the marked analogy which, I submit, exists also between chronic adhesive processes and arthritis deformans.

---

*Read before the Sections of Laryngology and Otology, at the Eighty-first Annual Meeting of the British Medical Association, held at Brighton, July, 1913.

Lucae[3] has asserted that it is not possible to separate otosclerosis and adhesive catarrhal processes clinically, but Politzer[4] differs from this view. Most present day otologists seem to be of the same opinion as Politzer, but many of the fuller descriptions of otosclerosis to be found still unavoidably mix up several of the main features of these two chronic ear affections, as is done in chronic diseases of joints.

The following are the headings under which points of analogy between otosclerosis and arthritis deformans are shown: Definition, etiology, course of disease, symmetry, trophic changes, morbid anatomy, pathogeny, and lines of treatment. Bearing upon etiology, the influence of age, sex, heredity, race, climate and other diseases and conditions, which are said to act as exciting causes of onset or exacerbations, are considered, and finally certain conclusions will be set forth.

DEFINITION.

Otosclerosis.—Politzer calls otosclerosis a progressive form of deafness, running its course without any catarrhal symptoms and presenting an entirely different character from the adhesive processes secondary to catarrh.

Kerrison gives the following definition: "The term otosclerosis is employed to describe the condition in which, independently of the health or intercurrent disease of the tympanum, the bony capsule surrounding the labyrinth is the seat of chronic nonsuppurative disease interfering with the function of hearing."

Arthritis Deformans.—Osler[5] describes it as a chronic disease of joints, characterized by changes in the synovial membranes and periarticular tissues, and in some cases by atrophic and hypertrophic changes in bones.

Keen[6] refers to it as a progressive nonsuppurative disease of joints, manifested by pain, swelling and impaired function, and leading to stiffness and deformity.

Llewellyn-Jones,[7] describing osteoarthritis, says it is a chronic oligoarticular disease of joints, the widely distributed form of which is most common in women, insidious in onset, running a slow, progressive course, the subjects of the disease being usually well nourished.

AGE.

Otosclerosis.—Gray[8] says that it is most frequent in middle life, rare under twenty, the majority of cases occurring between twenty and forty.

The commonest age incidence, according to Milligan,[9] is between fifteen and thirty.

Politzer states that it occurs especially often in elderly people, often in young healthy individuals, but those with a hereditary taint have symptoms marked at ten to fifteen.

Lake[10] gives the greatest incidence as occurring in young adult life, and states that it may begin even in childhood.

Arthritis Deformans.—The majority of Osler and McCrae's cases occurred between twenty and forty approximately. Half their cases began before the age of thirty years.

Llewellyn-Jones gives twenty to thirty as the period of greatest age incidence for rheumatoid arthritis, and says that osteoarthritis is commonest in middle and late life, but occurs in young females and even in children.

The majority of Garrod's[11] cases of arthritis deformans occurred between twenty and forty.

Painter says that rheumatoid arthritis is a disease of youth and middle life, and osteoarthritis of middle and late adult life.

SEX.

Otosclerosis.—Gray, quoting Bezold and Denker's combined cases, gives 60 per cent as occurring in women. Pierce[12] gives 60 per cent also, but Yearsley's[13] cases were as high as 83 per cent in females.

Politzer, Lake and Milligan state that females are more commonly affected than males.

Arthritis Deformans.—Painter says that osteoarthritis is more common in women than in men, but is more crippling in men, as the smaller joints are affected in women.

In Garrod's 500 cases of arthritis deformans, women were affected in 82 per cent.

In Llewellyn-Jones' 240 cases of rheumatoid arthritis, 83 per cent occurred in women. He also states that osteoarthritis is more common in females than in males.

Osler and McCrae, as well as Stewart,[14] give 50 per cent as the incidence in women.

HEREDITY.

Otosclerosis.—Bezold[15] found a history of heredity in 52, Denker[16] in 40.5, Siebenmann[17] in 35, and Pierce in 71 per cent of their cases.

Körner and Hammerschlag give a genealogic tree of five families in which forty-three cases of otosclerosis occur.

Arthritis Deformans.—Garrod's five hundred cases gave a family history of joint troubles in 43 per cent.

Osler's one hundred and seventy cases gave the same in 33 per cent, and he states that two or three children in a family may be affected.

RACE.

Neumann states that otosclerosis is much more common in the white race than in the negro, and Osler makes a similar statement regarding racial incidence about arthritis deformans.

CLIMATE AND CLIMATIC CONDITIONS.

Dryness and warmth are said to favorably influence the course of the disease in rheumatoid arthritis and chronic adhesive processes, and dampness and cold produce an injurious effect. With the exception of the benefit derived from being out of doors, climate has no influence over otosclerosis or osteoarthritis.

COURSE OF THE DISEASE.

Otosclerosis.—Lake, Milligan, Gray and Politzer say that the onset may be slow or rapid, the course progressive, though sometimes stationary for years, but may be arrested spontaneously with a fair degree of hearing.

Arthritis Deformans.—The onset may be slow or rapid, the course progressive, but in many cases, after involving two or three joints, the disease becomes arrested and no further development occurs.

SYMMETRY.

Both are usually bilateral, but one side is more advanced than the other in the slowly progressive cases. In the swiftly progressive forms early symmetry is the rule.

TROPHIC CHANGES.

Otosclerosis.—Lake gives the following: The external meatus is often large, due to change in the subepithelial tissues; the lining skin is polished, pale and devoid of cerumen; the eustachian tubes are large, with or without rhinitis sicca, the latter being frequently associated with otosclerosis. Less labyrinthine fluids or their entire absence in cases of long duration.

Politzer also mentions atrophy of the membrane. and the pale, dry condition of the external meatus, devoid of secretion.

Arthritis Deformans.—Changes in the skin and subcutaneous tissues, loss of hair, alteration in the nails, early localized sweats, followed later by cessation of this function over the affected areas, sensory and calorific disorders, wasting of muscles, cartilage and bone.

The trophic changes just enumerated are grouped under arthritis deformans by Osler and others, but Painter and Llewellyn-Jones seem to associate them exclusively with rheumatoid arthritis.

In many long standing cases of chronic hyperplastic otitis media, one finds a glossy, thin, flaccid, transparent membrane, through which the details of the inner wall of the cavum tympani can be discerned: the eustachian tubes are wide and free, and these are sometimes accompanied by a large, smooth, dry meatus, devoid of cerumen.

Are these trophic phenomena peculiar to chronic hyperplastic otitis media or to otosclerosis or to both? This question cannot be conclusively answered at present, but I think it is probable that cases of typical otosclerosis, like those of osteoarthritis, are not accompanied by any special trophic changes.

MORBID ANATOMY.

Otosclerosis.—Lake states that the earliest known change is a mucoperiostitis of the inner wall of the cavum tympani. In the cases in which the terminal lesions were examined, any or all of the following changes were found: The annular ligament had disappeared and was replaced by spongy bone, the cartilage which normally covers the footplate of the stapes and surrounds the oval window had disappeared and was replaced by new bone. Patches of osteoporosis occurred in the labyrinthine capsule, osteophytes were found in the oval window, some of which were intratympanic and others intralabyrinthine.

Gray says that the disease begins by the absorption of bone, and the region most commonly affected is supposed to be above and in front of the oval window, and that the changes may occur in any part of the temporal bone or the ossicles or the bones of the head.

Yearsley points out that the annular ligament is widest at its anterior superior margin and that the greatest degree of movement occurs here, and that perhaps irritation and friction may be a determining cause of the greater incidence at this situation. It is interesting to note in this connection that Painter lays special emphasis on the greater incidence of osteoarthritis in the joints most commonly used by athletes.

Gray further describes areas of bone which undergo absorption by osteoclasts, and the deposition of new bone by osteoblasts, the affected areas being sharply differentiated from the normal bone. The annular ligament and cartilage disappear and are replaced by spongy bone which shows a strong affinity for staining reagents, this bone becoming dense later.

Arthritis Deformans.—The following is a summary of the morbid changes occurring in osteoarthritis as described by Painter:

The synovial membrane shows practically no change, except about spurs which form at the junction of bone and cartilage. There is no diffuse infiltration of the capsule, as in rheumatoid arthritis. The cartilage hypertrophies at the edges, spurs form by the ossification of cartilage, whose cells are arranged in columns like those of rickets, at the junction of diaphyses and epiphyses, and these spurs become dense bone later.

The pathologic changes observed by Painter in rheumatoid arthritis may be summarized as follows: The synovial membranes are infiltrated and thickened, villi form and may ossify. The involvement of cartilage is next in order of frequency, but first in the order of clinical significance. It undergoes thinning, loss of sheen, striation and erosion. The bone is last affected, there is loss of calcium content, the intratrabecular tissue is replaced by fat and fibrous tissue, the trabeculæ stand out on section and are smaller and fewer in number. There is no evidence of necrosis.

Osler's description of the morbid anatomy of arthritis deformans is briefly as follows: The synovial membranes are thickened and fringes form, the periarticular tissues are infiltrated and swollen, the cartilage becomes soft and absorbed, or may disappear and be replaced by fibrous tissue of bone. The bone ends become smooth and eburnated in long standing cases, and the shafts atrophy.

New bone may form in the ligaments, especially in the spine, and may be followed by bony ankylosis, the latter occurring commonly in the vertebral joints, but rarely in those of the extremities. Muscles undergo rapid atrophy, and a radiograph shows atrophy and hypertrophy of bone and erosion of cartilage.

Ironside Bruce[18] points out that in early cases of osteoarthritis, areas of translucency, sharply marked off from normal bone, can be seen by the X-rays.

It will be observed that Painter gives the pathologic changes of rheumatoid and osteoarthritis separately, whilst Osler gives them combined under the title arthritis deformans.

In rheumatoid arthritis the soft tissues undergo early proliferation, to be followed by atrophy, which also involves cartilage and bone. Hoffa states that bony proliferations, when they do occur in rheumatoid arthritis, are in proportion to the amount of atrophy that has taken place. and he would regard them as reparative. (Llewellyn-Jones.) In osteoarthritis the soft tissues are practically unaffected, but cartilage proliferates and goes on to ossification, with the formation of osteophytes. It has been observed that the new bone formation in cases of otosclerosis is more than sufficient to replace the old bone and cartilage which has disappeared, and no explanation is offered to account for this excess of new growth.

In my opinion the excess of new bone formation, as represented by osteophytes in any joint, is due to the proliferation and hypertrophy of cartilage at certain points, this hypertrophied cartilage becoming ultimately ossified.

The microscopic pathologic changes given by Gray in his lucid description of the morbid anatomy of otosclerosis are practically identical with the physiologic phenomena of ossification in any cartilage developing bone, from the osteoclastic and osteoblastic activity down to the greater affinity of the new bone for staining reagents.

DISEASES AND CONDITIONS SAID TO ACT AS EXCITING CAUSES OF ONSET AND EXACERBATIONS.

The following list of diseases and other conditions have been mentioned as causes of onset and exacerbations in both affections: Syphilis, tuberculosis, gout, rheumatism, rickets, anemia, tonsillitis, exposure to cold and chill, inflamed processes in the nose, nasopharynx and middle ear, puerperium, pregnancy and lactation, anxiety, sorrow, depression and nursing the sick, excess of alcohol and tobacco, trauma and arteriosclerosis.

PATHOGENY.

The exact pathogenesis of otosclerosis and arthritis deformans is unknown, but many causse have been suggested, and these may be conveniently arranged under infective, nervous, vascular, metabolic and traumatic theories of origin, any or all of which may act by producing nutritional disturbances.

OTOSCLEROSIS.

Infective Theory (microbic).—Inflammatory middle ear processes (Katz,[19] Habermann,[20] Scheibe,[21] Ballenger[22]).

Primary osteitis of the labyrinth capsule which is independent of middle ear disease (Politzer).

Syphilis, supported by Habermann, but disputed by Korner. Congenital syphilis is considered by Lake to influence onset.

Toxic.—Stucky, quoted by Kerrison, suggests that toxins resulting from errors of diet and circulating in the blood are important factors in the progress of the disease, if not in its causation.

Apparently against the infective view is the observation put forward by Gray, Politzer and Moos,[23] that otosclerosis occurs without any trace of middle ear inflammation, and that of Gradenigo as to its negative bacteriology.

Nervous Theory.—Anxiety, sorrow and depression have been suggested by Walb.[24] Walb also states that there is a greater tendency for otosclerosis to develop in individuals who are subject to mental affections and nervousness.

Vascular Theory.—Arteriosclerosis as a cause in some cases is suggested by Maupetit.[25] Habermann found the foci of the disease in the temporal bone, grouped around the blood vessels entering from the periosteum of the tympanum. Anemia as a factor has been mentioned by Dundas Grant[26] and Gray, and the latter also describes areas of necrosis.

Metabolic Theory.—Puerperium, pregnancy and lactation. Bezold found otosclerosis associated in its onset with the puerperium in 18 per cent of one hundred and ninety cases. Politzer, Habermann and others have made similar observations.

Rickets (Politzer), gout (Tonybee, Buck and Argnano[27]).

Ossification of cartilage cells around labyrinth capsule (Siebenmann).

Part of a general sclerosis of the submucous tissue of the whole respiratory tract (Lake).

Traumatic Theory.—Sudden explosions, or injuries to the cranium, may increase or cause sudden deafness in these cases (Politzer).

ARTHRITIS DEFORMANS.

Infective Theory (microbic).—Organisms found in joints (Bannatyne,[28] Poynton and Payne.[29] Occurrence of influenza and gonorrhea as antecedent affections. Onset soon after tonsillitis (Bloodgood,[30] Llewellyn-Jones, Bannatyne).

Apparently against this view are the observations of Painter and Odery Symes,[31] who were unable to find organisms in the majority of the affected joints which were examined by them.

Toxic.—A toxic trophoneurosis (Tessier[32]), an autotoxemia (Tubby[33]), a cerebrospinal toxemia (Llewellyn-Jones).

Oral and alimentary sepsis have been suggested as the commonest source of the various types of toxemias.

Nervous Theory.—Worry, fatigue and depressing mental conditions. Changes found in the anterior cornua of the spinal cord (Folli,[34] Mott,[35] Bannatyne, Latham[36]). Lesions found in the peripheral nerves of joints (Pitres and Carriere[37]). The nervous theory is said by Stengel[38] and others to explain the articular changes, the muscular atrophy and the trophic disturbances in the skin.

Vascular Theory.—Arteriosclerosis is mentioned as a cause of osteoarthritis by Billroth,[39] Tessier and Lancereaux. Anemia is said to be present at the onset or during the course of the disease in osteoarthritis. Goldthwait is of opinion that the blood in osteoarthritis shows no special changes, and this view seems to be supported by the observations of Erving,[41] who made a careful blood examination in twenty cases affected by the disease.

I am not aware that this point has been fully tested in cases of otosclerosis whose appearance indicate the condition of anemia.

Metabolic Theory.—Pregnancy, lactation, puerperium and the menopause, thyroid, ovarian and uterine disturbances. Loss of calcium content in the bones, and the greater output than intake of calcium salts during the active period of the disease in rheumatoid arthritis (Painter). That the disease is due to a condition of acidosis (Barr,[42] King[43]).

Traumatic Theory.—Godlee[44] states that a severe injury in youth may determine an attack of osteoarthritis later in life. Painter points out the frequency of osteoarthritis in the most commonly used joints of athletes.

LINES OF TREATMENT.

The same or equivalent lines of treatment have been suggested in both affections.

Otosclerosis.—

Drugs.—Pot. iod., iron, arsenic, thyroid and thymus extracts, phosphorus, mercury.

Injections.—Fibrolysin, vaccines, intratympanic oils, etc.

Mechanical and Surgical.—Pneumomassage, probe massage, stapedectomy.

Counter Irritation.—Blistering, etc.

Arthritis Deformans.—

Drugs.—Pot. iod., iron, arsenic, thyroid and thymus extracts, etc.

Injections.—Fibrolysin, vaccines, intraarticular injections of sterilized vaselin (Buedinger[45]).

Mechanical and Surgical.—Massage, arthrectomy, cheilotomy (Handley and Ball[46]).

Counter Irritation.—Cautery, blistering.

CONCLUSIONS.

The following are the conclusions at which I have arrived:

1. That chronic adhesive processes of the ear, or, as Kerrison calls it, chronic hyperplastic otitis media, is rheumatoid arthritis affecting the organ of hearing.

2. That otosclerosis is osteoarthritis of the stapediovestibular articulation and of the bony labyrinthine capsule.

Dundas Grant has noted the frequent association of the two diseases in the same individual, and the occurrence of osteoarthritis in the female progenitor of many of the subjects of otosclerosis.

3. That the morbid changes of chronic hyperplastic otitis media and rheumatoid arthritis are practically identical, and those found in typical otosclerosis are similar to those found in osteoarthritis and spondylitis deformans.

4. That as all the affected structures are developmentally, anatomically and physiologically alike, and when in these structures identical types of destructive and reparative changes occur, the natural inference to be drawn is that these processes are the result of a common pathogenesis.

It may be urged that it is incorrect to try and establish an analogy between other articulations and that of the stapes in the oval window, but I submit that the analogy is complete from every standpoint. The periotic portion of the temporal bone is developed in cartilage around the auditory capsule, and in this respect is similar to other cartilage bones. Anatomically, the similarity is also complete. In ordinary joints of the extremities we find bone covered by hyalin cartilage, and when a joint cavity exists, synovial fluid is present. Synovial fluid, according to Halliburton, is viscid, rich lymph plus mucinoid material. It is secreted by the cells lining the synovial membrane, and its viscidity is due to the presence of mucin. In the absence of a joint cavity the bony skeletal elements, for the purpose of greater stability, are linked together by fibrous tissue or fibrocartilage, as in the case of the vertebral column in the mid-dorsal or lumbar region. (There are small synovial cavities in the cervical region between the lateral portions of the bodies.) The stapediovestibular joint is similar to a mid-dorsal articulation. The osseous labyrinth is a cartilage developed bone, and hyalin cartilage is found round the margin of the oval window and on the edge and vestibular aspect of the footplate of the stapes. The bony labyrinth walls contain interglobular cavities enclosing remnants of cartilage (Yearsley, quoting Manasse.)

The annular ligament is a fibrous structure which holds the stapes in position in the oval window. The perilymph, according to Halliburton, is a viscid fluid, not pure lymph, as it contains mucin, which is the cause of its viscidity. Physiologically, a common joint possesses the double function of being a sense organ of relative position and an organ of locomotion.

The labyrinth contains a sense organ of relative position for the head and the organ of hearing, the latter depending for the conduction of sounds on a system of joints.

5. That when, in addition to the identity of morbid anatomy, we find a striking analogy between the two diseases in the points of age, sex, race, hereditary incidence, symmetry of lesion, trophic changes, course, associated diseases and conditions which are said to influence onset and exacerbations, pathogeny and lines of treatment, the above seems to me to be the only conclusion to which an unbiased observer can come.

6. That in all the morbid changes there are two outstanding features, namely, atrophy and hypertrophy, running side by side or alternating, the atrophy being an active process as well as the hypertrophy.

7. That in the case of chronic hyperplastic otitis media and rheumatoid arthritis we find the most marked activity of the disease in the soft or clothing tissues, which first undergo thickening, to be frequently followed by atrophy, the latter ultimately involving the supporting structures.

8. That in the case of otosclerosis and osteoarthritis the most marked activity of the pathologic changes is in the firm or supporting tissues, the cartilage undergoing proliferation and ossification, with the formation of osteophytic overgrowths. The juxtaposition of hyalin cartilage and bone appears to offer the most favorable anatomic site of election at which these morbid processes take place.

If this view is correct, the included remnants of cartilage described by Manasse as being present in the osseous labyrinthine wall afford the necessary juxtaposition of the two tissues, and would explain the incidence of the patches of otosclerotic disease found scattered throughout the petrous portion of the temporal bone.

9. That the essential pathogenic factor underlying an active atrophy and hypertrophy can be only one, namely, nutritional, although the conditions which may cause nutritive disturbances are manifold.

10. That as nutritional disturbances in tissues can be brought about by infective, vascular, nervous, traumatic and metabolic influences, either or all of which may be acting in any given case, it is, therefore, impossible to fit otosclerosis and osteoarthritis into one airtight, pathogenic compartment.

I am, however, of the opinion that the essential factor underlying the morbid changes is a chemical one, affecting the nutritive stability of developing and fully developed bone and cartilage, comparable to the rachitic phenomena which occur in developing osseous tissues, but as to how these chemical disturbances are actually initiated, it is at present not possible to explain.

REFERENCES.

1. Painter: Amer. Pract, of Surg., 1907.
2. Kerrison: Diseases of the Ear, 1913.
3. Lucae: Progressiv. Schwerhörigkeit, Berlin, 1907.
4. Politzer: Diseases of the Ear, fifth edition.
5. Osler: Principles and Pract. of Med.
6. Keen: Textbook of Surgery.
7. Llewellyn-Jones: Arthritis Deformans, 1909.
8. Gray: Diseases of the Ear, 1910. Trans. Otolog. Soc., Vol. 7, 1906.
9. Milligan: Textbook of Diseases of the Ear.
10. Lake: Diseases of the Ear, fourth edition.
11. Garrod: Allbutt's System of Med., Vol. 3, second edition.
12. Pierce: Quoted by Yearsley.
13. Yearsley: Textbook of Diseases of the Ear, 1908.
14. Stewart: Quoted by Osler.
15. Bezold: Die Funktionel Prüf. d. menschl. Gehör.
16. Denker: Die Otosclerose.
17. Siebenmann: Zeitschr. f. Ohrenh., Bd. 34.
18. Bruce, Ironside: Quoted by Llewellyn-Jones.
19. Katz: Arch. f. Ohrenh., Bd. 53, S. 68.
20. Habermann: Arch. f. Ohrenh., Bd. 60, S. 37.
21. Scheibe: Verhandl. d. Deutsch. Otolog. Gesellsch, 1901.
22. Ballenger: Textbook of Diseases of Ear, Nose and Throat.
23. Moos: Quoted by Gray.
24. Walb: Schwartze, Handbuch d. Ohrenh.
25. Maupetit: These de Bordeaux, 1905.
26. Grant, Dundas: Trans. Otolog. Soc., Vol. 7, 1906.
27. Argnano: Rev. Hebd. de Laryn., 1896.
28. Bannatyne: Rheumatoid Arthritis.
29. Poynton and Payne: Trans. Path. Soc., Part 2, 1902.
30. Bloodgood: Progress. Med., Vol. 4, 1907.
31. Symes, Odery: Rheumatic Diseases.
32. Tessier: Rheumatismes, 1908.
33. Tubby: Lancet, December 26, 1908.
34. Folli: 11 Policlinico, December, 1904.
35. Mott: Braine, 1902.
36. Latham: Lancet, April 6, 1903.
37. Pitres and Carriere: Arch. General de Bordeaux, 1898.
38. Stengel: Amer. Jour. Med. Sciences, 1903.
39. Billroth: Surgery, New Sydenham Soc. Transl.
40. Goldthwait: Boston Med. and Surg. Jour., 1904.
41. Erving: Amer. Jour. Med. Sciences, 1903.
42. Barr: Brit. Med. Jour., April, 1913.
43. King: Johns Hopkins Bulletin, 1907.
44. Godlee: Roy. Soc. Med., Clin. Sect., March, 1908.
45. Buedinger: Quoted by Tubby.
46. Handley and Ball: Brit. Med. Jour., May 13. 1913.

LX.

COMPLICATIONS THAT MAY ARISE DURING OR AFTER THE SUBMUCOUS OPERATION FOR THE CORRECTION OF SEPTAL DEVIATIONS.

By George W. Mackenzie, M. D.,

Philadelphia.

The operation referred to is the submucous resection after Killian, occasionally modified, but slightly, according to the indications in individual cases.

It is only after an experience of eight years, during which time about 386 cases have been operated upon, that I feel justified in writing upon the subject. I might add, too, the experience obtained in controlling student physicians in their attempts at the operation.

To me the most dreaded complication is infection, the prevention of which prompts me to outline briefly the technic employed in preparing the tables, instruments and patient, together with a description of the operation.

All instruments but the sharp cutting ones are collected and put into the sterilizer, together with a small enamel cup which is to hold the Schleich's solution. The scalpel, the swivel knife and the needle of the infiltrating syringe are put into a separate tray containing lysol solution or alcohol. Two glass-topped tables are cleansed, first with bichlorid solution (1 to 500) and then with 95 per cent alcohol. Towels are not used. The table on the operator's right hand side is to hold the instruments, the table on the left is for cotton, gauze strips, Schleich's solution, and the instruments for their administration, together with one nasal forcep to be used by the assistant in mopping or supplying mops to be used by the operator.

The patient is directed to wash his face thoroughly with soap and water, with particular attention paid to the region of the mouth and nose, after which the face is gently mopped with alcohol. The patient's collar is loosened, a long sterilized rubber apron is put on, and the head covered with a sterile cap. He is then directed to sit on the chair between the two tables. The operator and assistant put on short sleeved sterile coats or gowns. They then scrub up thoroughly for five minutes with pumice soap and warm water, followed by a dip in alcohol. The head mirror is mopped off with alcohol, using caution to prevent the solution from getting through to the under surface of the mirror, after having once ruined a mirror by immersion in alcohol.

The assistant cuts a one inch sterilized gauze bandage into strips about eight or ten inches long, to be used as mops, and later for dressings. The nurse mixes the Schleich's solution, a powder containing morphin muriate .005, cocain. mur. 0.1, sodii chlorid 0.2, which is dissolved in 100 cc. of hot sterile water to which is added ten drops of adrenalin (1 to 1000). After two or three preliminary applications of 20 per cent cocain are made, at intervals of three minutes, the injection of Schleich's solution is begun, using a Reiner or Record syringe (capacity thirty minims), fitted with a fairly large bored reinforced needle. The first syringeful is injected submucously, as far back on one side as possible, followed by a second, 1.5 cm. above, below or in front of the first. At the beginning the solution goes into the tissues very slowly, but gradually it goes easier. Both sides are injected until all of the mucous membrane is infiltrated and the mucoperichondrium and mucoperiosteum are fairly loosened from the underlying cartilage or bone. By injecting the solution thoroughly, I have frequently been able to loosen the periosteum from even sharp ridges. In the case of ridges it is well to inject thoroughly both above and below them. The fact is the injection, if well done, partly accomplishes the first step of the operation. Injections are made all the way anteriorly to the tip of the nose, occasionally swelling the soft tissues of the tip, making the nose appear pudgy. I am convinced that proper infiltration makes the operation less painful and considerably easier to perform than the mere application of a local anesthetic to the mucous membrane. It also is a factor in reducing the length of time necessary for the performance of the operation.

The nurse takes her position behind the patient, to steady and support the head and to move it into various positions as directed by the operator. A blunt aneurism needle, which I have been accustomed to use as a hook, is introduced into the right or left nostril, depending upon the side from which the operation is to be performed. The nurse holds the hook by the upper extremity of the handle, in such a manner as to prevent her hand from coming in contact with the surgeon's or interfering in any way with his movements.

When operating from the left side the index finger of the surgeon's left hand is inserted into the right nostril, pushing the anterior portion of the cartilage to the left, while the thumb of the same hand pulls or twists the softer tissues to the right. This exposes the anterior edge of the quadrangular cartilage to the best possible view on the left side; while, at the same time, the mucous membrane is drawn forward 4 or 5 mm. from where it normally belongs; thus, an incision made through the mucous membrane at or near the edge of the cartilage falls naturally 4 or 5 mm. behind the anterior edge of the cartilage. Here I follow the plan of attempting to leave a narrow strip (2 or 3 mm.) of the anterior edge of the cartilage intact.

For the incision I use a short, wide-bellied Murphy knife, and endeavor to go through the mucoperichondrium with the first attempt, but more often it requires three or four. I do not find it necessary to curve the incision, as many operators do. Where the incision, in spite of my efforts, is too short, I lengthen it, after partial separation with the raspatory. A speculum is placed into the left side and the nostril dilated vertically, using the blades of the speculum to protect the nose against undue injury with the knife. With the speculum in place, and the assistant holding the raspatory in the pocket between the mucous membrane and the cartilage, at the same time using traction downward and lateralward, bringing the upper end of the wound considerably downward, I am enabled, with the cutting edge of the knife directed upward and medianward, to lengthen the incision 2 or 3 mm. Then changing the direction of traction, the incision is lengthened downward. The blades of the speculum stop the knife from cutting the skin on the inner surface of the nostril, an accident that is liable to occur without the use of the speculum in this way.

With the Killian raspatory (double end) the mucous membrane is separated from the cartilage and bone to or beyond all the deviated parts. In fact, if the infiltration has been thorough, it is possible to go over the apex of sharp pointed spines and spurs without perforating the mucous membrane; of course, not always. After thorough preparation of one side the other is begun. To reach the opposite side the same Murphy knife is used to make the incision through the cartilage down to the mucous membrane of the right side, but not through it. The index finger and thumb of the left hand are applied after the same manner as was done in beginning the first incision. This dislocates the edge of the cartilage posterior to the incision to the left, bringing it into the full view of the operator, who, with the raspatory, begins to separate the cartilage from the perichondrium of the right side, following up the separation as thoroughly as on the left side. When the mucous membrane of both sides is fairly well separated the ordinary Hajek speculum is introduced, one blade on the left side between the mucoperichondrium and the cartilage, and the other blade on the right side in a similar manner. If any further separation of the mucous membrane is desired and possible, it may be attempted while the speculum is thus held. The next step is the use of the swivel knife. If the knife is at all dull, or if the cartilage tends to buckle under pressure of the knife, the incision is started with sharp pointed scissors, and followed up with the swivel knife. The instrument is pushed in an upward and posterior direction as far as it will readily go; occasionally, when the septum is deviated considerably in its anterior portion, it is necessary to tilt the knife laterally in a direction corresponding to the inclination of the cartilage. The knife is then carried in a curved direction (convexity posteriorly) downward as far as it will go without risk of engaging and tearing the mucous membrane. It is then brought forward; when, as ordinarily occurs, a fair sized (4 or 5 sq. cm.) piece of cartilage is cut loose. This is removed with a rat tooth forceps or any other suitable instrument while the speculum still remains in position. The effect of removing a large piece of cartilage at the first attempt is somewhat dramatic to one observing the performance, although the removal of a smaller piece at one time is attended, perhaps, with less risk of tearing the mucous membrane. I frequently go back a second and occasionally a third time with the swivel knife for a strip of cartilage before using the biting forceps. From this stage on I rarely find it necessary to remove the speculum. If necessary, I mop with a gauze strip, one end of which is fed into the opening between the blades of the speculum, pushing it in firmly with the raspatory.

Further preparation of the mucous membrane of both sides is made as the necessity of the case may require. Removal of all deviated bone with bone cutting forceps (Middleton) is attempted; in some cases even to the posterior edge of the septum, and in others to the floor of the nose. I find that by keeping at it I can succeed in dissecting the inferior edge of the cartilage from the groove of the vomer with which it articulates. Finally, the mucous membrane is prepared from the upper thickened portions of the vomer on both sides. Then with a dovetailed chisel a goodly portion of it is chiseled loose, when its removal is completed with an alligator or other form of forceps.

I do not feel satisfied with my work until I am able to see the posterior pharyngeal wall through one side as freely as through the other. When the deflection requires it, I am liberal in removing the upper anterior portion of the septum; otherwise not.

At this stage of the operation the speculum is removed and the patient is directed to blow the nose somewhat forcibly; first one side, then the other, to clear out any excess of blood that may prevent me from obtaining a good view of the entire field. Besides, the effect of forcibly blowing the nose is to replace any accidentally torn or folded pieces of mucous membrane. I find that slight tearing of the mucous membrane occasionally happens with the most expert operators, and its occurrence should cause no feeling of embarrassment. I have repeatedly observed that its occurrence does not delay the recovery or cause any inconvenience to the patient, provided it is recognized and the flap properly replaced before applying the dressings. In fact, a small tear will in a measure assist drainage. In some cases I have gone even so far as to make deliberate punctures of the mucous membrane at the completion of the operation, where they have not occurred accidentally, for the purpose of affording better drainage, and with good results. The cases selected for the purpose have usually been those which I have been compelled to operate on high up, where, because of the difficulty of packing, the risk of hematoma is increased. I do not mean to infer that I am accustomed to making accidental tears; on the contrary, it is a rare occurrence.

Before dressing the case a careful inspection of the pocket is made, after first thoroughly mopping it out with gauze, to ascertain if any loose fragments of bone remain. After satisfying myself on this point and making sure that every torn piece of mucous membrane has been properly replaced, the dressings are applied. Strips of one inch sterile gauze are carefully packed into each nasal fossa, sufficiently snug to hold the raw surfaces in close contact.

The incision wound is not sewed together. An outer dressing of gauze is applied, and over all adhesive strips to hold them in place.

I do not claim that the method here outlined is original, nor do I claim it to be superior to any of the other methods of performing the submucous operation, but the observations to be described concerning the complications and the methods employed to prevent and treat them are original.

Cocain or adrenalin poisoning may occur during the preliminary cocainization of the mucous membrane, or during the infiltration. Cocain may produce various and contradictory symptoms. I have noticed that they occur most frequently among those who were previously nervous and anxious. The symptom most commonly observed is an increase of a preexisting nervousness. The patient develops a paleness of the face and trembling of the extremities, and, when most pronounced, the patient is liable to faint; at the same time the pupils dilate slightly. It is often difficult, at least for the writer, to separate the cocain symptoms in nervous patients from the psychic disturbances that might have occurred had plain sterile water been used. I find by putting the patient on a couch in a recumbent position and reassuring him, that the symptoms pass over promptly. Alcoholics, particularly, develop loquacity and boastfulness, combined with fear. I have never observed any poisoning effect from cocain that I could truly classify as alarming. Occasionally vomiting occurs. In these cases the pulse is somewhat quickened and tension diminished. Summary of symptoms includes pallor of face and lips, tremors, fainting, dilatation of the pupils, rapid and feeble pulse, vomiting, loquacity, anxiety, followed later (five to ten hours) by headache. I may add that amyl nitrite by inhalation is a rather effectual antidote to cocain poisoning.

There is another group of symptoms different from those already mentioned occurring, but rarely, from the adrenalin that has been added to the Schleich's solution. The symptoms observed have been rather opposite to those produced by cocain. The patient complains of throbbing sensations in the whole body, but especially of the heart. He shows symptoms of anxiety; the face becomes somewhat cyanotic and the pulse fairly rapid and of very high tension. These symptoms are of short duration (from one to two minutes), but recur with each injection of the solution. By changing to a fresh Schleich's solution minus the adrenalin, the symptoms do not reappear.

I have observed that cases which could not tolerate the least amount of adrenalin do very well on Schleich's solution alone. I might add that a careful chest examination in those susceptible to adrenalin has frequently revealed some lesion of the heart.

There is still another complication that might occur during the infiltration of Schleich's solution, of which I wish to speak, and that is air embolism. My first experience was the most startling. I had been using a syringe that was not in perfect working order. The piston was unduly loose in the barrel. As a result, it would occasionally suck in air, in spite of my efforts to avoid it. On this particular occasion about a cubic centimeter was injected, when, some seconds later, the patient developed diplopia with marked vertigo, vomiting, and some other less important nervous phenomena. The patient was put on a couch and a casual observation revealed a white tree-like formation on one side of his face, corresponding to the outlines of the facial artery and its several branches. The patient's condition improved after a half hour; however, some effects remained for two or three days. Evidently the air had gotten into the circulation through the puncture of a vein. A few years later I had a second similar experience, but fortunately the effects were less severe. I felt reasonably certain that the second case did not get any air, but of this fact I cannot be sure, for the effects corresponded, so far as the white tree formation, to that of the first case. Following close on the second experience a third occurred. This time an assistant was injecting the solution into another physician, preparatory to operation. This case showed the same tree formation plus a ptosis and slight paresis of the superior rectus eye muscle, all on the right side. The effects in this case passed off entirely in about one hour. Just how many other cases may have had air emboli in other parts of the body, without apparent symptoms, cannot be determined. My object in citing these unpleasant experiences is to point out the possibility of their occurrence to those who have not yet experienced them, and to caution the beginner against the risk he takes in not seeing to it that the syringe, after loading, is free of air before injection. Furthermore, the risk of air embolism is greater when using a syringe that is not in perfect working order than one that is.

The next possible complication—or, perhaps better put, embarrassment—occurs from an incomplete or improper primary incision of the mucous membrane. It is essential that the first incision be made down to the cartilage and sufficiently long, for if not, the subsequent attempt at separation of the perichondrium will be futile.

I have watched beginners work for a half hour or more trying to separate the perichondrium, and lacerate the mucous membrane considerably because of their failure to cut clean through the perichondrium with the primary incision. I am speaking also from personal experience when I, too, was a beginner, so that I feel justified in emphasizing the importance of the first incision. If the incision has been made sufficiently deep and sufficiently long, it will not be necessary to lengthen it backward along the floor. An incision along the floor increases the risk of tearing the mucous membrane in this region. A horizontal running incision is prone to start a tear that is difficult to control. In a general way, a perforation or a tear accidentally made is of no great importance so long as the surgeon is aware of it. The only danger lies in not recognizing it immediately, for when not recognized the surgeon invariably keeps enlarging it and adding to his trouble. A very good practice is to have the patient blow the nose frequently, as this tends to replace flaps into their normal position.

After separating the mucoperichondrium in good shape for a centimeter or so, it occasionally happens that the surgeon is balked. He seems to be unable to progress farther. If he attempts to force things, he perforates, as a rule, the same, but occasionally the other side. In those cases where the raspatory perforates the same side, the apparent obstruction has been a socalled adhesion; a condition where the perichondrium is more or less bound down to the cartilage, due, perhaps, to some old inflammatory process. Besides being more adherent, the membrane at this point is thinner and less resistant and tears readily. This difficulty might have been anticipated and guarded against by a more thorough infiltration at this particular location. During the infiltration of the septal mucous membrane I am in the habit of looking out for locations where the infiltration appears to be more difficult, and give to them more particular attention. I do not cease my efforts until the mucous membrane is found to be quite free of the cartilage or bone throughout. When infiltrating in a location obscure from view—for instance, under the ledge of a pronounced spur—the ease or difficulty of infiltration is decided by the sense of touch conveyed through the point of the needle, or by the resistance offered to the infiltration, as told by the pressure necessary to empty the syringe, or by the amount of solution that leaks from the nose when the patient puts his head forward, as he is instructed to do after each injection. If only half the quantity is lost by dripping from the nose, at least some has been retained.

In the case where the raspatory perforates the opposite side, the cause is due either to lack of experience or more often to a preexisting fracture with overlapping edges. For instance, if the posterior edge of the short anterior portion is inclined to the right, and the overlapping anterior edge of the larger posterior part to the left, it is easy to comprehend how, when operating from the left side, the raspatory, after encountering some temporary resistance, will naturally follow the plane of the anterior portion and come out on the right side of the nose. It would not be fair to term such a performance an act of clumsiness. The best means to guard against this complication is to avoid undue forward pressure with the raspatory the moment the obstacle is met. Stop and proceed to the next step of the operation in the same manner as though this obstacle had not been present. With the knife incise the cartilage, following the same direction and length of incision as the primary one—through the cartilage, but not through the mucoperichondrium of the right side. If working from the left side, use the thumb and index finger of the left hand to dislocate the cut edges of the cartilage, and proceed after the manner already described. While separating the mucoperichondrium of the right side, when the site corresponding to the obstruction is reached, it will be found, by hugging the cartilage closely, that the instrument will jump over the posterior edge of the anterior fractured part without much difficulty. Separate the perichondrium freely in upward and downward directions, and be contented for the time being. Withdraw the instruments and proceed with the next step, the same as you would have done had no complication occurred. With an ordinary speculum, introduce one blade between the cartilage and perichondrium on one side, and the other blade between the cartilage and perichondrium on the opposite side, so that when the blades are separated the mucous membrane is protected by the blades of the speculum on each side and the cartilage is free in the middle. Push the speculum forward (into the nose) as far as it will go, without undue pressure or risk of tearing the mucous membrane. Remove, with swivel knife, scissors or punch or what you will, all of the cartilage that can be removed without injuring the mucous membrane. By so doing one will be able to remove quite easily all of the cartilage anterior to the fracture, including the overlapping portion that inclined to the right. At the completion of this step one is able to see quite clearly the right side of the remaining cartilage posteriorly, more or less free of mucous membrane, and it will be noted, too, that the raspatory follows the right surface with ease. In our earlier experience we are not able to tell immediately just where we are. We look for a free edge of the cartilage, and are surprised at not finding it. With closer inspection and by feeling with the raspatory, the inferior edge of the cartilage may be found hugging the left side of the mucoperichondrium and blending with it. This condition has been brought about by the productive (inflammatory) connective tissue, the result of the healing process that followed the original fracture, perhaps years before. After more or less manipulation with the raspatory, one is able to get between the inferior edge of the fracture and the mucoperichondrium of the left side at one point or another, when further dissection becomes quite easy.

Perhaps the most frequent complication which occurs more or less often in the experience of most of us is perforation of the mucous membrane. Unintentional perforations may occur from lack of experience (a stage through which we must all pass before we become expert); or to undue haste or recklessness, for which there is no excuse; or to certain mechanical difficulties which make a perforation nigh impossible to avert. For lack of experience the only advice is practice; more preferably on the cadaver, but where the cadaver cannot be secured, on living subjects under the guidance of an expert. For recklessness there is no advice. For mechanical difficulties the best scheme is to anticipate them and by proper infiltration try to reduce them to a minimum. The less our experience the more we are inclined to attribute our difficulties to unavoidable causes. Experience, together with proper infiltration, tends, therefore, to reduce the complications, both in number and magnitude.

One or two buttonholes in the mucous membrane need cause no anxiety, provided two of them do not occur at corresponding points of the two sides, when they are liable to be followed by a through and through perforation. A recognized buttonhole is not nearly so important a complication as one unrecognized; for, when not recognized, the operator is liable to enlarge it unintentionally. I have watched students and assistants work away trying to get past a buttonhole for a long time without success. In fact, I have been told by certain honest friends in the profession that they have worked under such conditions for a long time without success, and in disgust have given up further efforts. For this reason I feel warranted in pointing out a simple way to pull oneself out of such an embarrassment.

If for any reason a perforation has occurred during the separation of the mucous membrane from the cartilage, and the operator is sufficiently experienced to recognize it, he will find it a wise procedure not to attempt further separation at this point. Try further separation at a point above or below the perforation and get well beyond it, and then work toward the perforation from above and posteriorly, or from below and posteriorly. By so doing one avoids the risk of enlarging the perforation and at the same time avoids loss of time. Do not attempt to remove cartilage or bone with septal forceps until a sufficiently large area about the part to be removed is separated from the mucous membrane, the object being to avoid the possibility of grasping any part of the mucous membrane with the forceps. If the operator, in spite of his efforts to avoid it, should grasp the mucous membrane at any point, he will know it when he attempts to bite or fracture off a piece of cartilage or bone and remove it. A piece of cartilage or bone free of the mucous membrane should come out easily without any resistance. If there is resistance, he may look for one of two things: either he has too large a piece of bone with a pointed edge engaged in the mucous membrane, when force is likely to cut or tear the mucous membrane during its removal; or else he has a piece of the mucous membrane within the grasp of his forceps. In the first instance a rotation in one or the other direction causes the sharp edge of bone to free itself from the mucous membrane, when the piece comes out easily. In the second instance, cease pulling, or else a good sized piece of mucous membrane will be pulled out with the bone. Remove the forceps and try to separate the mucous membrane more thoroughly, after which you may try again. If necessary, repeat this performance several times, until you are able to bite off a fragment without engaging the mucous membrane in the forceps.

Occasionally it occurs that all but the apex of a sharp spur is free, and this holds tightly to the mucous membrane, preventing easy removal.

Other physical causes that lead to tearing of the mucous membrane, after it has been freely prepared from the cartilage and bone, is the use of dovetailed chisels; especially, when chiseling in an oblique direction, as we are compelled to do in some instances. This was not an infrequent cause with me in my earlier experience. To avoid it I now shave off smaller pieces and am satisfied to go back several times, instead of attempting to chisel off too much at one time. I may mention, too, that in using the swivel knife one is liable (in certain cases where it is necessary to push the knife in an oblique direction) to engage the mucous membrane with the protruding side of the instrument and thus go through it. This error is likely to occur when one is overambitious to remove a large first piece of cartilage. It is a mistake for an operator to attempt any display of this kind, for his pride is likely to have a fall, when he is compelled to work overtime on the balance of the operation.

Another less frequent cause of perforation is due to faulty separation of the periosteum from the sides of the articular edge of the vomer, especially in cases where the bone is thickened (osseous spine). At this point the periosteum runs up from below and follows the superior edge and down the other side, as one more or less continuous layer. When separating the mucoperichondrium from the cartilage, it will be noted that the separation progresses readily until a point is reached inferiorly, corresponding to the superior edge of the vomer. Here the raspatory balks, and if sudden force is used the membrane tears. It is never so tight at this point, however, but that milder efforts applied in the proper direction and repeated often enough will result in separation with an intact mucous membrane. A little care with sufficient patience is the only skill required to accomplish the result. Another method to overcome the difficulty is to remove all of the cartilage first, leaving only the superior or grooved edge of the vomer; then with a scalpel cut through the periosteum along the superior margin of the bone, and through this incision gradually work a separation of the periosteum from the vomer.

Excessive bleeding during the operation may be a complication of sufficient importance to cause some timid operators to abandon further efforts at the operation. Excessive bleedings are usually venous. In some cases bleeding occurs so profuse that the blood literally pours out of the nose. In other instances, though less pronounced, it nevertheless is sufficient to well up in the pocket between the layers of mucous membrane, and thus hamper the operator in getting a proper view of the field of work. The bleeding is due generally to an injury of a vein coursing near the floor of the nose. For this reason I would suggest a plan that I have long since adopted, namely, to leave the work to be done along the floor until the last, so that if by any mischance a hemorrhage should occur, let it do so after every other part has been taken care of.

Since operating in a field of blood is more or less unsafe, it is well to know just what to do under the circumstance. The plan that has usually succeeded in stilling the bleeding, in my experience, has been the tight packing of the cavity with sterile gauze for a few minutes or longer. I have found it occasionally necessary to use in addition a little adrenalin. After the bleeding has been stilled it sometimes recurs when we attempt to take up our work again. A second or even a third pause may be necessary before we can complete the operation. In some rare cases I have completed the work satisfactorily in spite of the presence of the blood, depending upon the sense of touch; however, I do not recommend this procedure. When the operation is completed I do not let the hemorrhage worry me, even though it is quite marked, for I have found that proper packing of the nose, as regularly practiced, to replace and hold the two apposing surfaces of mucous membrane together, is sufficient to control the bleeding. Though not doubting its possibility, I have never witnessed a secondary hemorrhage, therefore cannot speak of it from experience. Slight oozing occurs in all cases after the submucous operation.

Retention of a fragment of bone or cartilage.—Lack of precaution to remove all loose fragments of bone that may have fallen into the bottom of the pocket, or that may have been unintentionally tucked away between the remaining intact bone and periosteum, is liable to lead to complications later. Though I have never known a piece to be subsequently thrown off, it is possible that such a thing might occur if the retained piece is sufficiently large. In at least one case, I believe that a long lasting localized and firm thickening of the septum, quite far back, was due to a retained fragment of bone. I would emphasize, therefore, the importance of carefully mopping out the wound cavity of blood and searching for small fragments of bone. In my earlier experience I am sure that pieces of bone must occasionally have been retained, from the fact that since having exercised extra precautions, I have been surprised to find how often I was able to detect fragments of bone located in obscure places.

Here, I am reminded of an error that I have committed on more than one occasion. It can hardly be classed as a complication, yet it has prevented me from obtaining as perfect results as were anticipated. It is a fracture of the septum near the posterior extremity. It occurs when one side has been thoroughly prepared and the other not, combined with the improper use of the septal forceps; as, for instance, using the forceps to break the bone instead of biting it off. If we should grasp a portion of the bone that had been only partially prepared and use pressure in a lateral direction, the bone will break at the point of the instrument; and a second fracture is also likely to occur posterior to this, but in the opposite direction. Since the posterior fragment is united with the periosteum on one side, it is retained, but in a distorted position. Subsequent healing of this second fracture in a faulty position may cause a narrowing on one side near the posterior nares. On the second or third visit after removal of the dressings, an angular deformity may be observed near the posterior nares. This is fairly easy to correct by resetting the fractured bone in a more favorable position. It is better practice, however, to avoid such a fracture by using proper precautions in manipulating the septal forceps.

Faulty packing of the nose with gauze may be responsible for complications after operation. For instance, if a piece of gauze hangs down into the pharynx from the posterior nares, the patient may be bothered with a sensation of a foreign body in the pharynx, causing him to hawk to get rid of it; or if low enough to touch the base of the tongue, may cause a gagging sensation and vomiting, or even fits of spasmodic coughing. After having one experience of this kind I have guarded against it since. I found the patient quite nervous and fearful of choking. The tip of the gauze was grasped with a nasal forceps introduced into the pharynx and promptly removed without difficulty, and to the delight of the patient. Since that experience I carefully examine all cases by posterior rhinoscopy, to see that none of the gauze protrudes into the pharynx. In packing the nose I have always used several short strips (eight to ten inches long) rather than one much longer, so that in the event of a piece getting too far posteriorly it can be removed without disturbing the whole dressing.

Faulty packing of the gauze into the nose may result in getting a part of the gauze into or through a perforation and between the layers of mucous membrane, with the result that prompt healing is prevented. I have seen this occur twice, but not from my own packing. To avoid this I have the patient blow his nose—first one side, then the other—rather forcibly at the completion of the operation, and again just before packing. With a suitable speculum, a long one is safer, introduced in such a manner that one of the blades holds the flap in place, begin to pack the nose. Insert the first strip of gauze between the speculum blades (separated as wide as the space permits), along the floor of the nose from the posterior to the anterior nares. With the nasal or dressing forceps applied over the gauze, using pressure downward to hold the gauze in place, the speculum is withdrawn. Then shift the pressure of the forceps applied to the gauze in a median direction, to cover and protect the tear, and while holding it there reintroduce the speculum; but this time with the median blade directed against the piece of gauze already in place, at the same time withdrawing the forceps. Introduce a second strip of gauze after the same manner as the first, and so on until this side of the nose is fairly well packed. The other side may be packed in a similar manner. In the average case I find that two or three pieces of one inch gauze, ten inches long, on each side, suffices for a dressing. Some cases require more and others less. Faulty packing may also result in the tucking of a torn flap inward on itself, so that more or less of the mucous surface of the flap lies directly against the raw surface of the mucoperichondrium of the opposite side. Its prevention is the same as that just described in packing of the nose in cases of perforations. If either of these complications—i. e., gauze between the layers of the mucous membrane of the two sides or tucking inward of a torn flap—be discovered after the removal of the dressings, it can be corrected by proper replacement of the flap and then holding it in position, for two or three days, with properly applied gauze.

I cannot speak of complications arising from other methods of dressing the nose, since I have had no experience with them. I have not yet attempted the method of no packing, but am tempted to at some future time, selecting a case with complicating suppurative sinusitis, where packing appears to me to be more or less contraindicated.

A very unusual complication that occurred in the experience of the writer was that of breaking a chisel in the patient's nose. The case was one with a bilateral ridge, causing the upper portion of the vomer to be very broad. A medium sized dovetailed chisel was used. A larger one would have been better. A few firm strokes with a metal mallet drove the chisel into the bone for about a half inch. Since the bone did not yield, an attempt was made to withdraw the instrument and try at another place. As it did not come out readily, a twisting movement was made, with the unfortunate result that the chisel blade broke off flush with the bone at its point of entrance. I then attempted to remove thinner pieces, shaving the bone off, so to speak, until the broken chisel was reached. In spite of my precaution a second chisel was broken in the same way as the first. The result was that we had two pieces of chisel wedged fast in the bone, one a few millimeters above the other. A third attempt with another chisel was successful, both in removing the broad bone (thin layers at a time) and the recovery of the broken fragments of the other two chisels. My mistake was in attempting to take off too large a piece of very broad and hard bone, and in using a medium instead of a large sized chisel. Another error was that of attempting to twist the chisel to break off the bone when the bone was more resistant than the chisel. I cite this incident, hoping thereby that someone else might be saved a similar embarrassment. Aside from delaying the operation, no untoward after-effects resulted.

We shall next consider the complications that may occur after the submucous operation, but before doing so I would like to tell briefly just what I do, and advise the patient what to do at the completion of the operation.

After the gauze has been packed into the nose in the manner already described, strips of plain gauze are folded to make a pad of eight or ten thicknesses, one inch wide and about two and one-half inches long. This is put over the horizontal portion of the external nose and folded up on the two sides, and over all zinc oxid adhesive strips are used to hold the pad in place.

The patient is then told that there will occur an oozing of blood which will gradually change to a bloody serum, diminishing in amount until it ceases altogether in about twenty-four hours, and the total amount will probably not exceed two or three fluid ounces. The patient is told to expect a slight headache the evening of the operation, and not to worry about it should it occur. The contributory causes of the headache are the use of cocain, the slight shock of the operation, and the pressure of the pack in the nose. They are also instructed to keep quiet and go on a cool liquid diet for at least twenty-four hours. In other words, even though the patient may feel good, it is better that he be treated as though he was actually sick, for by so doing he may be prevented from becoming sick. The patient is further told not to disturb the dressing nor let anyone else do so without instructions, to take no stimulants and to sleep the first night with the head high, and finally he is given aconite, one drop of the tincture every two hours, for two days. As a rule these patients do not sleep well the first night, but they do the second.

It is unusual for a patient to have a rise of temperature. When it does occur, it rarely exceeds 99.4°. In some cases the dressings are removed in twenty-four hours, in others forty-eight hours. In cases where there has been a pre-existing subacute or chronic sinusitis, the dressings are removed in twenty-four hours, when a careful inspection is made. A redressing is never applied unless hemorrhage should occur, and then only when it persists. Persistence of bleeding for ten or fifteen minutes after removal of the first dressing, calls for a pack to control it. This pack of sterile gauze is applied directly to the bleeding point. To avoid this secondary hemorrhage the gauze of the first dressing should be removed slowly until it is noticed that resistance is offered, and with the resistance bleeding. The gauze removed up to this point is cut off flush with the nose, and the remaining gauze allowed to remain in place until the following day, when it will be found to come out easily and without risk of bleeding.

If the case has done normally, after removal of the gauze dressing a small piece of sterile cotton is applied into the side of the nose where the incision had been made, and worn for a day or so to protect the wound from dust and dirt. The patient is usually able to return to his regular occupation three or four days after the operation. A normal appearing case is one which, after the removal of the dressings, shows no sign of reaction—i. e., the mucous membrane is neither too red nor swollen. In brief, the mucous membrane should appear about the same as before the operation. If the mucous membrane of the septum is red, swollen and succulent in appearance, it is an indication of infection, and the degree of the infection is in proportion to the intensity of these signs. A slight amount of swelling clears up in a few days; a greater degree requires a longer time. In exceptional cases (in my experience, about one and a half per cent) the swelling may last two or three weeks. In these more intense cases of swelling of the septum, excessive reaction, there is a like swelling of the mucous membrane of the entire nasal cavity, and with it a seromucous discharge accompanied with fever (100° to 101°), stoppage of the nose and headache. In some of these cases I have suspected an abscess of the septum, and accordingly, after an application of cocain and adrenalin, have incised the swelling freely on both sides (making in all four or five incisions, a half inch long) without, to my surprise, obtaining pus. Some of the cases thus treated were apparently helped, while others showed no improvement whatever. In all but one of the cases showing this excessive reaction there followed a depression of the nose. I have come, therefore, to consider this complication (inflammatory reaction) as one likely to be followed by another, namely, depression of the bridge of the nose below the inferior margin of the nasal bones.

In my experience the factor that has contributed more than any other to the production of this intense reaction has been that of combining with the submucous resection an operation on one of the turbinates, especially the middle. The nasal cavity will tolerate packing fairly well, so long as there has been no particular bruising or laceration of the mucous membrane; but let there be gross injuries to the mucous membrane, for instance, lacerations, especially when they are not properly replaced; or let there be a fair sized piece of turbinate removed, leaving a large raw surface, then will packing the nose be likely to be followed by intense reaction. It is better, therefore, when doing the submucous resection, not to attempt at the same sitting any other operation on the nose, unless it is particularly indicated or of vital importance, in which event it would be well to avoid packing the nose. Infection follows operative wounds of the turbinates with decidedly greater frequency where the nose has been packed than where it has not. I have seen these infections of the turbinate so intense and widespread as to involve the entire nasal cavity. In fact, the infection of a wound of a turbinate may spread to one or more of the sinuses, producing an empyema. So, too, we must not forget the possibility of an overlooked or latent empyema infecting a wound of the turbinate.

Another source of infection following the submucous operation is injury to the septal mucous membrane in a case where there already exists an empyema of one or more of the accessory sinuses. Here the gauze as commonly used for packing after the submucous operation bridges the way, so to speak, for the infection to travel from the exit of the infected sinus to the wound on the septum. Though I have been fortunate enough never to have had an infection following the septum operation, severe enough to cause intracranial complications and death, I can appreciate the possibility of its occurrence.

Following the submucous operation there occurs occasionally a circumscribed bilateral swelling of the septum. This swelling is more frequently due to a hematoma, i. e., an accumulation of blood between two layers of the mucous membrane. A hematoma may occur at any location, but more often in the upper anterior portion; due, no doubt, to the difficulty encountered here in applying the dressings. This complication is more or less preventable by seeing to it that the dressings are applied in such a manner as to bring a fair pressure over the entire operated portion of the septum. Another preventive measure is to puncture the septum at two or more places on each side, at the completion of the operation and before applying the dressing.

The treatment of a hematoma following the septum operation is a free incision of both sides, taking care, however, that the two incisions are not directly opposite one another, because of the risk of a permanent perforation. If the swelling is a real hematoma, it will be noted that a dark, almost black, liquid blood will issue from one or more of the incisions. The issue of blood from the incision is facilitated by the patient blowing the nose rather forcefully. Pressure and counterpressure upon opposite sides of the septum may be tried; but I have not found it necessary, since the squeezing process fails to express any additional blood after the nose has been freely blown.

A common complication of overestimated importance is a permanent perforation. The fear of it has deterred many a beginner from attempting the submucous operation sooner. By way of encouragement to him who is overanxious about this complication, I would advise that he have less fear; for it is far better to have the operation followed by a perforation, than to allow a badly deflected septum to go without operation. I have come to be somewhat liberal after having had the experience of seeing six of my own cases result in a permanent perforation; otherwise, recovery in every one of these cases was prompt and perfect. One of the troublesome features of permanent perforations, when small, is a whistling sound produced by hard breathing and blowing of the nose. This may become corrected later, as the perforation tends somewhat to enlarge spontaneously. An irregular or angular perforation becomes round or oval shaped. Should the whistling noise persist, then the perforation may be enlarged by the surgeon. A case in which the mucous membrane has been torn at corresponding points on both sides, one, therefore, with a tendency to a permanent through and through perforation, is less liable to develop a hematoma or infection than a case in which a perforation has not occurred, since these perforations afford effectual means for drainage. This feature was recognized by Johann Fein, who took advantage of it and deliberately made a liberal incision on both sides with the object of obtaining a permanent perforation. Whether he still continues the practice, I cannot say.

Erysipelas is a complication about as prone to follow septum operations as any other operation. One such case was seen by the writer while doing postgraduate work. This case I recall occurred in a patient who, after his operation, sat and conversed with another patient infected with erysipelas in the waiting room of a clinic. I never learned what happened to this patient eventually, for he was transferred to a hospital for infectious diseases.

Some authors doubt the possibility of the transferability of erysipelas. Without debating the point at this time, I believe its possibility is sufficient to warrant us in isolating such cases. To avoid the risk of erysipelas and other infections, patients are instructed to keep to themselves in an atmosphere as free as possible from dust. It is well also to postpone operation of the septum in a case with acutely infected tonsils or adenoids. This is a point upon which too much stress cannot be laid. In a case with enlarged tonsils and with the history of frequent attacks of tonsillitis, though the tonsils at the time may not appear acutely inflamed, a pack in the nose, since it compels mouth breathing temporarily, is liable to awaken an acute attack of tonsillitis from which focus infection may spread to the septum. The same applies, with even greater tendency, to the adenoids. There are patients with enlarged adenoids, who are subject to attacks of acute inflammation similar to that which occurs in the tonsils, while the tonsils may be quite insignificant. Such an adenoid can cause an infection of the septum as effectually as the tonsils; therefore, in all cases, we should inspect both tonsils and adenoids first, and when found to be enlarged or otherwise affected, they should be removed before attempting the submucous resection of the septum.

Acute empyema of one or more of the accessory sinuses is a complication that may follow in the wake of a septum operation, and, therefore, deserves consideration. Some of the cases suspected as being acute empyema following the septum operation may be acute recurrences of unrecognized chronic empyema. In the cases seen by the writer, however, the evidence pointed to their being primary acute attacks. I am reminded here of a case with severe frontal headaches on the right side, with a pronounced deviation high up, associated with polyps and pus in abundance. I decided to operate to make room for subsequent work in the region of the middle meatus of the right side. The patient had no complaint to make of the left side; accordingly, my whole attention was centered on the right side. A septum operation combined with free polyp extraction on the right side was followed by marked relief, but on the third day the patient developed pain in the region of the left frontal sinus. Careful inspection showed signs of acute and active empyema of that sinus, which lasted two or three weeks before it entirely cleared up. The occurrence of this complication following the submucous resection does not exceed 2 per cent or 3 per cent. The precautions I would suggest against this possible complication are to avoid operation during an attack of acute "cold in the head," remove diseased and enlarged tonsils and adenoids previously, and avoid combining other operations with the submucous. In some cases I have postponed operation upon the septum for several weeks. I do not mean to infer that I would delay operation upon the septum too long in the face of a severe attack of sinusitis, for there are some few cases where the operation upon the septum may be the very thing to make room for drainage, and thus terminate the attack of sinusitis, as happened in this case on the right side.

The complication—or perhaps better, the undesirable after-effect of the submucous resection—that has grieved me the most of any, has been depression of the nose between the lower edge of the nasal bone and the tip of the nose, causing it to look somewhat flattened from the front view. Though I have been able to observe it more or less in at least 5 per cent of the cases operated (fortunately for my comfort), but one patient spoke to me of it afterward, and he was so incensed that he advised three of my prospective patients against the operation. In at least one other case—viz., one with a pronounced deviation combined with sinus disease and polyps, requiring several mechanical sittings—the patient as well as myself observed that the nose which was inclined to the left before the operation was inclined more afterward. This patient was so gratified at the results otherwise obtained that she did not put up much of an objection.

Besides infection after operation, the removal of the cartilage too far forward may act as a cause in producing this deformity. The experienced surgeon is more liable to this fault—i. e., the removal of too much cartilage anteriorly—than the beginner, from the fact that with experience we are usually more thorough and bold. There are some cases where the deviation is so pronounced anteriorly that the most favorable results, so far as breathing and ventilation are concerned, can be obtained only by the more thorough removal of the cartilage, with the risk of deformity. In those cases where the edge of the cartilage facing forward is quite straight, it is unnecessary to remove as much of it as in other cases where it is deviated.

In submucous septum operations it is my practice to remove all of the bone and cartilage that is deflected, but no more.

Infections and hematomas contribute in a measure to external deformities, and should be avoided, if possible, by takink precautions after the manner previously suggested. Relative to infections, it may be suggested by some that cleansing the nose with an antiseptic wash be tried in order to prevent it. My reply to such a criticism is that an antiseptic to be strong enough to kill bacteria would be strong enough to injure the vitality of the epithelium, the secretion of which is normally antiseptic; and by using the antiseptic, this protective barrier is removed and a gateway to infection is opened that otherwise would have remained closed. I believe that severe infections occur more frequently when antiseptics, i. e., antiseptics in the true sense of the word, are used, than when not used.

Concerning the operation in cases that have been previously attempted, there is something to be said, since in a measure they come to us as more or less complicated cases.

One of the best authorities, Hajek, up to a few years ago, and possibly to this day, advised strongly against operating on a septum that had been previously bungled by someone else; and the more so when there has been a resulting perforation. He claimed that it is well-nigh impossible to operate them successfully. Notwithstanding his admonition, I have operated several such cases with perhaps a little less ease than others, but with just as satisfactory results. If one observes the precautions outlined in this paper concerning thorough infiltration with Schleich's solution, and the method of going past a perforation by a sort of circuitous route, he can surely accomplish his purpose. In one of the cases operated, where the nose seemed to have been literally battered up, I was able to get through with the work nicely, to my astonishment, and without even attempting it, the result was a complete closure of the perforation.

SUMMARY.

1. Operations should not be performed in the presence of acute rhinitis, tonsillitis or inflammation of the adenoids.

2. If the patient presents enlarged tonsils or adenoids, with the history of acute attacks of tonsillitis, and especially peritonsillar abscess, the tonsils should first be removed before attempting the submucous operation.

3. Operating on the septum in the presence of an acute or subacute emypema of any of the accessory sinuses should be avoided unless the patient's condition necessitates the operation for the purpose of securing drainage.

4. The previous failure of some one else, even though the

case presents a perforation, need not deter anyone with sufficient skill and courage from attempting the operation.

5. Some system of technic that will insure the least possible risk of infection from without should be adopted.

6. Strong antiseptic washes should not be used in the nose, before or after operating the septum.

7. Thorough infiltration of the mucous membrane gives more intense and longer lasting anesthesia than the local applications alone.

8. Thorough infiltration partly prepares the mucous membrane from the cartilage and bone, and thus shortens the length of the operation. It also thickens the mucous membrane so that it is less liable to tear.

9. Cocain poisoning seldom occurs, and when it does, it passes off promptly. Adrenalin poisoning occurs even less frequently, and passes off just as promptly.

10. The primary incision should be made with a sharp knife, not too far back, and as long as the space will permit. It need not be curved or made angular shaped.

11. Avoid, if possible, lacerating the mucous membrane, and if, in spite of precautions, a tear should occur, do not let it worry you overmuch.

12. In the event of a tear high up, plus an empyema, it would be better not to use gauze dressings, for in that region the gauze will act as a carrier to convey the pus to the wound.

13. All crooked portions of the septal cartilage and bone should be removed.

14. The removal of too much cartilage anteriorly and high up, when this portion of the septum is fairly straight, should be avoided.

15. In using the chisel near the floor of the nose, small pieces should be removed rather than to attempt too much, because of the risk of breaking off the chisel in the hard bone.

16. In the event of an unintentional fracture far back, the fractured portion should be removed, or if it is not denuded enough to do so, on account of one side being attached, the fracture should be reduced.

17. In packing the nose the gauze or whatever is used should be applied with even, fairly firm pressure over the entire mucous membrane of the two sides, corresponding to the area from which the bone and cartilage has been removed.

18. After operation the patient should be given explicit directions as to what he is to do and what not to do. In fact he should be treated in as strict a manner as one would after any other operation of like magnitude.

19. After removing the dressings, a bilateral diffuse swelling, with some fever, indicates an infection, which is to be treated accordingly.

20. A bilateral circumscribed swelling of the septum speaks for a hematoma, and should be freely incised from both sides.

21. Where a through and through perforation is found after operation, it should be left alone. If it is small and the patient finds a whistle in his nose, the perforation may be enlarged.

22. A rise of temperature with pain or headache calls for removal of dressings, for the probability is that there is an infection, and the sooner the dressings are removed the less severe the infection is likely to be.

23. Too much should not be attempted at one sitting. If the submucous operation has been performed, additional work upon the turbinates should be postponed. Removal of polyps may occasionally be attempted.

24. If some deformity occurs after the operation, in spite of every precaution, the only thing that can be done is to trust that the patient's improvement otherwise overbalances it. Apropos of the subject of slight deformities, let all conscientious surgeons be liberal enough to defend one another.

In conclusion, I wish to say, that in spite of all its drawbacks, which when put together appear so immense, I believe, sincerely, that the submucous operation of the septum is the most uniformly satisfactory operation that we as rhinologists are called upon to perform.

LXI.

APHTHOUS ULCERATION OF THE UPPER AIR PASSAGES IN PULMONARY TUBERCULOSIS—A RETROSPECT AND A REVIEW.*

By John N. Mackenzie, M. D.,

Baltimore.

There is perhaps no subject in the whole domain of laryngeal pathology which has excited such universal interest and awakened so much animated discussion as the pathologic conception of laryngotracheal phthisis. The association of ulcerative disease of the upper air passages with pulmonary consumption has been recognized from an early time, and, although over a century has elapsed since the publication of Petit's little classic,[1] the most divergent opinions have prevailed concerning the correct interpretation of their reciprocal relationship. The older writers described only one form of ulceration, and busied themselves with the question of a laryngeal phthisis, in the generic sense of the term, without making those finer histologic distinctions which pathologic anatomy later developed. The existence of true tubercle as the chief etiologic factor in the production of what is commonly known as phthisis laryngea cannot be gainsaid, in the light of pathologic investigation, and it now only remains to be decided whether all forms of ulceration of the upper air passages which are encountered in the course of pulmonary tuberculosis are to be referred directly to the tubercle bacillus as their primary cause, or whether, on the other hand, there are not some which may be regarded as owing their origin to influences associated with, but not necessarily dependent upon, the true tubercular process.

I propose in this paper to consider a variety of ulceration which has an important bearing on this question, and which, on account of its more frequent localization in the lower portion of the trachea and bronchi, and its usual occurrence in the later stages of pulmonary tuberculosis, comes more within the notice of the pathologic anatomist than under the eye of the clinical observer. It is probably due mainly to this fact that it has been overlooked by many of the leading writers on diseases of the throat, notably in England and America, and its existence lost sight of in the discussion of more pressing and therefore more engrossing questions.

---

*Read before the American Laryngological Association, Atlantic City, N. J., May 9, 1912.

This, the socalled aphthous erosion of the older writers, was first depicted in the atlases of Cruveilhier[2] and Albers,[3] and clearly distinguished from tubercular ulceration by Rokitansky.[4] In his Lehrbuch der pathologischen Anatomie, Rokitansky calls attention to round or oval erosions of the mucous membrane, surrounded by a scarlet border and covered with a yellowish slough, which are found, during the course of pulmonary phthisis, in great numbers in the trachea, and which, after the extrusion of the slough, coalesce and lend to the membrane a sieve-like, worm-eaten appearance. These, which are not to be confounded with tubercular ulceration, are found, according to this observer, in greatest number on the side of the trachea corresponding to the lung whose disorganization is the more pronounced.

Föster,[5] who also clearly recognized this peculiar form of ulceration, whilst admitting its aphthous nature and pointing significantly to the fact that it may resemble tubercular ulceration so closely as to render an appeal to the microscope necessary to differentiate them, asserts as the result of his observations that the majority of the socalled aphthous erosions of the trachea are in reality tubercular. On the other hand, many authors look upon these ulcers as catarrhal (Louis,[6] Trousseau,[7] Andral,[8] Hasse,[9] Rheiner,[10] Rühle,[11], Heinze,[12] and others), or, as Rindfleisch has done, refer them to follicular ulceration; whilst their tubercular nature is maintained by Isambert,[13] Ziegler,[14] Eppinger,[15] and others. Eppinger, who describes them as tubercular arrosions, held, in the absence of absolute histologic proof of direct tubercular infiltration, that they are, notwithstanding, specific tubercular products, and suggested that they may be evoked through the operation of the monas tuberculosum of Klebs.

Many years ago I examined the respiratory organs of all patients dead of tuberculosis in the Rudolph and Jewish hospitals of Vienna during a period extending over five months, and published the results of the examination of this abundant material in the *Monatsschrift für Ohrenheilkunde*[16] in Berlin.[17] I wish today to present to you a description of this aphthous erosion, based upon an analysis of these cases, and to attempt to define, as accurately as possible, the relations between it and pulmonary tuberculosis.

Macroscopic Appearances.—Aphthous ulceration appears as small, for the most part superficial, irregularly round or oval erosion of the mucous membrane, which vary in size from a pin's head to a small pea, and which, separate and distinct, stud the surface of the membrane, or, coalescing, form a large ulcer, which spreads in an irregular manner over the mucous surface. Commencing as superficial erosions of the mucous membrane, with tolerably defined margins, they assume later a more characteristic appearance; their walls become sharp, irregular, and somewhat elevated, and vary in color from a light reddish shade to a pronounced livid hue. The base is either smooth and red in color, or presents a rough, uneven appearance. It is generally covered with a yellow slough, or with a more or less orange colored exudation. The ulcers are surrounded by a brilliant scarlet border, and the mucous membrane between them is slightly swollen and deeply hyperemic, thus forming a pathologic picture of great beauty. The ulcers, which are always multiple and never single, are most frequently met with, and are by far more abundant in the lower portion of the trachea and bronchi; in the former the posterior wall seems to be their most frequent seat, though the whole mucous membrane may be covered with them. I have seen the trachea in its entire length and circumference, together with the bronchi of both sides, almost completely destroyed and its mucous membrane converted into a necrotic mass. In my experience they are never found in the bronchi of the unaffected lung, but where both lungs are disorganized, the bronchi of both sides are studded with them, and in most cases they can be traced directly into the cavities themselves. They are met with less frequently in the larynx and the pharynx. In the former they select the laryngeal aspect of the epiglottis, the anterior surface of the arytenoid cartilages and the interarytenoid fold as their favorite seats. In the pharynx I have seen them most frequently in the pyriform sinuses, where they sometimes assume a considerable size. Their occurrence here is most probably explained by the accumulation in these cavities of the irritant sputum. In the larynx they lose to a great extent their characteristic appearances and approach more closely the type of simple catarrhal ulceration. They are sometimes found associated with or in the neighborhood of tubercular ulcers, and I have seen them more frequently than tubercular ulceration in the trachea.

Microscopic Appearances.—The sections presented in general the typical picture of circumscribed necrosis of the mucous membrane. At those situations where sloughs were present, the epithelium and upper layers of the mucosa were converted into a finely granular detritus, and the neighboring connective tissue of the mucosa, and also, to a great extent, the submucosa, were highly infiltrated with lymphoid cells. This round cell infiltration invaded the tissues between the mucous follicles, without, however, involving them in a marked degree in the inflammatory process. The spots where ulceration had already occurred presented in the base and edges of the ulcer likewise a granular detritus, and the surrounding tissues were also the seat of a high grade round cell infiltration. Here and there it was possible to recognize transition stages of both the above described processes; viz., nodules, so to speak, of necrobiotic tissue, in which the slough had already begun to separate, although adhering closely at several points. The examination of a great many sections showed that the first stage of the aphthous process consisted in a high grade round celled infiltration of the mucous membrane in its upper layers, with cloudy swelling of the superimposed epithelium. Distinct miliary tubercles were nowhere to be seen, neither within nor in the neighborhood of the necrobiotic nodules. Sometimes individual nodules coalesced, and especially was this true of the bronchi, so that the membrane over a considerable area presented the above described sloughing of the upper layers of the mucosa. The above anatomic appearances, therefore, leave no room for doubt that these ulcerated areas are the result of a circumscribed superficial mycotic or septic inflammation of the mucous membrane—that is to say, an infiltration of its tissues with so rich and rapid cell proliferation as to eventuate in necrosis and sloughing of the superficial layers.

In view, then, of this interpretation of their nature, what practical conclusions can be drawn concerning the etiology of these lesions? Are they related to the tubercular process in the lungs, and, if so, what is the nature of the relationship? The first question I must answer decidedly in the affirmative, since they are most frequently found in connection with well pronounced tubercular changes in the lungs; and in regard to the second, it seems impossible to resist the conclusion that the foul contents of the pulmonary caverns. lingering in the bronchi and lower trachea, so irritate the mucous membrane as to cause it to react with tissue necrosis.

It may be here remarked that in years gone by two opinions prevailed concerning the part which the sputum plays in the production of laryngeal phthisis. Following Louis,[18] the advocates of the one affirmed that the sputum and ulcers stand to each other in the relation of cause and effect, although they differed widely as to the nature of the ulcerative process, some maintaining it to be a simple catarrhal erosion, while others regarded it as tubercular, evoked through the invasion or infection, as it were, of the superficial layers of the mucosa by the micrococci in the expectorated matter (Ziegler, Eppinger). The champions of the other and perhaps more popular idea were equally confirmed in their belief that the sputum played no role whatsoever in the destructive changes, and some even went so far as to deny the possibility of corrosion of an intact mucous membrane by the contents of the pulmonary caverns (Heinze).

Passing then to a summary of the evidence upon which the corelationship between aphthous as contradistinguished from tubercular ulceration of the air passages and the corrosive action of the sputum rests, the following facts present themselves in favor of the view expressed above:

(a) The predilection of the ulcers for those places which are in constant contact with the sputum, viz., the trachea and the bronchi, and especially the posterior wall of the former and the laryngeal surface of the epiglottis.

(b) The fact that they increase in number as the lungs are approached, and that they are much less common in the upper portion of the trachea and larynx. It was these familiar facts that suggested to Louis the hypothesis which bears his name.

(c) That they may be traced from the bifurcation of the trachea to the division of the bronchial tubes where in all cases they become visible only in one tube, which is that leading to a cavity. This remarkable distribution of the ulceration in pulmonary tuberculosis, which I have repeatedly verified by dissection, did not escape the observation of Stokes, who advanced it in favor of Louis' hypothesis.

(d) Their absence in the bronchi of lungs which are not the seat of cavities or advanced tubercular change. This, at least, has been my observation, and I am unaware that they have been seen by others when the lungs were not the seat of pronounced disorganization.

(e) Their occasional presence in the esophagus, stomach and intestines from swallowing the sputum. In one of my cases ulceration was found in the esophagus and small intestine macro- and microscopically identical with the aphthous ulcer in the larynx. Tubercle was present only in the lungs.

(f) Finally, their occasional appearance in suppurative pneumonia (Wunderlich[19]) and gangrene of the lung.

These lesions, therefore, may be regarded as the result of an inoculation, so to speak, of the mucous membrane with the detritus from the broken down pulmonary tissue, leading to the formation of a loss of substance pathologically distinct from, but possessing some of the characteristics of, the tubercular ulcer. Notwithstanding, however, that these nodules owe their existence to the disintegration in the lung, it does not seem to me justifiable to look upon them, as Eppinger has done, as specific tubercular products, for the following reasons:

First—That they differ histologically in no respect from the circumscribed mycotic or septic ulceration of the mucous membranes of individuals in whom no evidence of tuberculosis exists; an important point in their differentiation from the well defined and characteristic anatomic changes of tubercular infiltration and ulceration. In this connection, it may be added that the tubercular process, as shown by Heinze, commences under the epithelium, whereas in the ulcers under review that structure is always first affected.

Second—That well pronounced circumscribed inflammatory infiltration resulting from necrobiosis and sloughing and histologically identical with these aphthous lesions is found, although not as frequently and as abundantly, in the mucous membrane of the air passages, and particularly in that of the trachea, as a secondary complication in diseases other than pulmonary tuberculosis. Thus, in circumscribed gangrene of the lung, or as the result of perforation into the trachea of broken down, sloughing bronchial glands, small, round "aphthous" erosions may be detected in the mucous membrane of the trachea and bronchi, which are the histologic analogues of those described above.

Third—That on other mucous membranes of the body, under similar conditions, namely, as the result of a neighboring long standing gangrenous process, or the constant passage over them of an ichorous discharge, ulceration is found, identical in every respect with that described above. This is true, for instance, of the mucous membrane of the vagina in gangrenous conditions of the uterus. In one of my cases of this kind the vagina was studded with small aphthous erosions, the result of gangrene of the portio vaginalis uteri from an unknown cause.

The great number of the ulcers, their absence in intense, acute and chronic simple catarrh of the pharyngobronchial membrane, their characteristic microscopic appearances, especially their often elevated, livid, irregular edges and sloughing base, and the picture which the microscope reveals, are sufficient to exclude the idea of their catarrhal nature, and strongly militate against the hypothesis which would relegate them to the simple inflammatory condition of the mucous membrane which often ushers in or accompanies the tubercular process in the air passages.

I think, therefore, in conclusion, that my observations go to show that aphthous ulceration of the larynx, trachea and bronchi occurring in the course of pulmonary tuberculosis is related to the affection in the lung in so far as it is produced by the corrosive action of the sputum; but that in the present state of our knowledge (1880) it is impossible to affirm its specific tubercular nature.

At the time these observations were made our study of the subject was rendered infinitely more difficult than it is at present by our comparatively scant knowledge of its bacteriology, and the technic of examination then in use was ridiculously primitive when compared with the methods of precision and exactness of modern scientific research. Moreover, the darkness surrounding the investigation of the disease had not been illumined by the isolation of the tubercle bacillus, and we had therefore to work out the problem without the light of that great discovery.

For these reasons it is not surprising that in days gone by there should have been so much divergence of opinion in regard to the ulceration under consideration. But even with modern histologic and bacteriologic methods of investigation there was still no consensus of opinion among authorities on the subject until the atmosphere of the question was cleared by the painstaking and interesting researches of Ziba, in the Pathological Institute at Strassburg.

Before referring to this important work permit me to pass briefly in retrospect and review the opinions of some of those who have specially investigated the subject histologically during the past thirty years, and who from the carefulness of their studies are entitled to eminent respect.

While Virchow[20] admitted the possibility of "erosive" ulceration of the larynx and trachea in tuberculosis, B. Fränkel,[21] on the other hand, maintained, from the presence of tubercle bacilli in the secretion obtained from them, that the erosions in question were true tubercular products. Schech,[22] while acknowledging that he was repeatedly unable to find either tubercle or bacilli in the walls and base of the ulcer, nevertheless declared that it is a specific tubercular lesion and sought to explain the frequent absence of characteristic structure and bacillus on the ground that at the time of the examination the tubercles had already been cast off or were lost to sight in the overwhelming multitude of the lymphoid cells of the secretion. Heryng,[23] as the result of numerous bacteriologic examinations, agrees with Schottelius that simple erosions of the mucous membrane of the larynx may exist which are due to the secretion from the lung, and which may occur quite apart from infection by the tubercle bacillus, but which can subsequently be secondarily infected with tuberculosis.

Orth[24] believes that the vast majority of the socalled "aphthous," "erosive" or "corrosive" losses of substance are true tubercular ulcers. At the same time he calls attention to the fact that the sputum of the cavities contain, often in considerable number, bacteria other than the tubercle bacillus, and that furthermore, chemical changes may occur in the secretion which may exert a corrosive or septic or necrotic influence quite aside from that produced by the specific poison of tuberculosis.

Eugen Fränkel[25] concludes from his histologic and bacteriologic researches that all destructive lesions of the larynx occurring during pulmonary tuberculosis are due to the operation of the tubercle bacillus, which invades the tissues through the epithelium, and is often assisted in its destructive action by pyogenic organisms. This observer admits, however, that there are some cases of ulceration which cannot be regarded as specific tubercular products, since they show neither the characteristic anatomic changes of the disease nor the presence of the bacillus. He would set these cases aside as examples of mycotic epithelium necrosis due to invasion of pathogenic cocci through the surface. This view of Fränkel that the overwhelming majority of larynx ulcers in consumption are true tubercular in origin is shared by most pathologists and laryngologists, with the exception perhaps of Gottstein,[26] who regards the socalled aphthous ulcer as the result of simple necrotic sloughing of the epithelium.

In talking the matter over with Professor Hans Chiari in the autumn of 1910, when he was in this country to deliver the Herter lectures at the Johns Hopkins University, he was very positive in his opinion that the work done by me over thirty years ago was good today; but that it would have to be gone over again with modern methods of examination, and in the light of the discovery of the tubercle bacillus; and he furthermore said that on his return to Strassburg he would have it done.

The following spring the work of Ziba,[27] inspired by Chiari, appeared, which, I venture to think, has shed much light upon the subject, and which is important from the fact that the author has approached the question in a different and more comprehensive and liberal way than that pursued by some other observers. For a detailed account of his observations and methods I must refer to the original publication, and content myself simply with a summary of his conclusions.

As the result of his histologic and bacteriologic researches he believes with me in the existence of the aphthous ulcer of the larynx and trachea in pulmonary tuberculosis, as contradistinguished from the true tubercular ulcer. He has found that the aphthous ulcer is caused by streptococci in the sputum from the lung cavities—that infection takes place from the inner surface of the mucosa and produces an inflammatory process which is associated with tissue necrosis, and which leads to the formation of the aphthous ulcer—the latter may later become tubercular, but in the aphthous ulcer originally there is neither in its base nor walls any trace of miliary tubercle. This conception of its nature is, furthermore, supported by bacteriologic examination. Only in those ulcers in which miliary tubercle was discovered microscopically were tubercle bacilli found, and solely in the region of the miliary tubercle itself. In all these cases both miliary tubercle and tubercle bacilli were absent. Even in those cases in which tubercle bacilli were found in the secretion which covered the mucous membrane, both characteristic structure and bacillus were wanting.

Aphthous ulceration of the larynx and trachea produced by streptococci may invite the development of tubercular ulceration. This is all the more probable when it is remembered that it appears in greatest numbers in the last stages of pulmonary tuberculosis. At this period the great majority of the ulcers are simply streptomycotic in character, and secondary tuberculosis develops in them only here and there.

I have brought this question before you for discussion reluctantly and with much diffidence on account of my personal interest in the matter. But, quite apart from any such consideration, the subject is deserving of attention in view of the fact that it is overlooked by nearly all English-speaking authors on tuberculosis of the upper air passages.

BIBLIOGRAPHY.

1. De phthisi laryngéa, Monspelier. 1790.
2. Livre 35, Pl. 4, Fig. 3.
3. II, Tafel 13, Fig. 4.
4. Lehrbuch d. pathol. Anatomie, Bd. III, Wien, 1861, S. 19.
5. Lehrbuch d. path. Anatomie, Jena, 1864, p. 312.
6. Recherches sur la phthisie, Paris, 1825.
7. Traité de la phthisie laryngée, Paris, 1827.
8. Clinique Médicale, Tome II, Paris, 1829.
9. Speciel. patholog. Anatomie, Bd. I, Leipzig, 1841.
10. Virchow's Archiv., V, 1853.
11. Die Kehlkopfkrankheiten, Berlin, 1861.
12. Die Kehlkopfschwindsucht, Leipzig, 1879.
13. Annales des maladies de l'oreille, etc., II, 3, p. 162, 1876.
14. Lehrbuch d. pathol. Gewebelehre, IV Auflag., Leipzig, 1875.
15. Volkmann's Vorlesungen. VI Serie, S. 1305.

16. Pathol. Anatomie des Larynx u. der Trachea, Berlin, 1880.

17. No. 9, 1881. Ueber die sogennanten aphthösen Substanzverluste auf der Schleimhaut des Larynx, der Trachea und der Bronchien bei tuberculöser Lungenphthisie. See also a paper in the Transactions of the Medical and Chirurgical Faculty of Maryland, 1882—Diphtheritic Ulceration of the Upper Air Passages and Its Relation to Pulmonary Phthisis.

18. Op. citat. This popular view was advanced over a century before the time of Louis, by Sylvius (Opera Medica, Trajenum ad Rhenum, 1695, p. 692). Vide Virchow, Die Krankhaften Geschwülste, 2, S. 644-645.

19. Handbuch d. Pathologie u. Therapie, III, 2, p. 96, Stuttgart, 1856.

20. Berlin. klin. Wochenschrift, 1883, pp. 109, 135.

21. Berlin. klin. Wochenschrift, 1883, No. 4, p. 523.

22. Volkmann's Sammlung, Series 8, 230, 1883. See also the chapter on tuberculosis of the larynx in Heymann's Handbuch der Laryngologie und Rhinologie, 1898.

23. Die Heilbarkeit d. Larynxphthisie und ihre chirurg. Behandlung, 1887.

24. Lehrbuch d. Spec. Pathologie, 1887, Bd. I.

25. Virchow's Archiv., 1890, No. 121.

26. Die Krankheiten des Kehlkopfs, 1893.

27. Fraenkel's Archiv. für Laryngologie und Rhinologie, 1911, Bd. XXIV, Heft 3.

LXII.

IMMEDIATE AND REMOTE SEQUELÆ OF RADICAL REMOVAL OF THE FAUCIAL TONSIL—OBSERVATION OF TWO HUNDRED CASES.*

By Virginius Dabney, M. D.,

Washington.

When we reflect that operation looking to the removal or reduction in size of the tonsil was devised and practiced in the middle ages, the amount of literature on this subject in all its phases is in nowise surprising, great though it be. Hence, a few only of those articles pertinent to the limited scope of this paper will be quoted here. Similarly, the facility with which any point of view could be sustained by reference to these authors is obvious, but it is one thing to state a fact and quite another to account scientifically for its existence. Thus, the statement of sterility in the male or female who has had a tonsillectomy done before puberty, the occurrence of blindness and other calamities of physical origin, all may have existed, but such post hoc, propter hoc reasoning cannot convict the laryngologist.

In order to narrow the field of this paper the radical removal alone is considered, which can, of course, be done by as many different instruments as methods; obviously, again, the partial and the radical method may share in common some of the sequelæ, notably hemorrhage and injury to the adnexa. Consequently, I do not wish to be understood as limiting any given result to the radical method merely because I mention it in that connection.

The agitation to stay the hand of the laryngologist in his alleged ruthless onslaught on the innocent tonsil is timely, if a little overdone, insofar as it has directed attention to the harmful effects that may and do follow the removal of these problematic organs. This plea for considerate surgery is eminently proper, but the lay press has made such spectacular use of it that a legitimate operation, even where it is vital to the patient's health, is now performed under great protest on the part of parents who have had their opinions moulded by the public prints.

---

*Candidate's thesis, American Laryngological Association.

PRIMARY AND SECONDARY HEMORRHAGE.

This sequela is given prime consideration by all observers, though a few seem to regard it as not especially serious (Freer, Richardson, Ballenger), from which view I must sharply dissent. Although I have never had nor seen a fatal hemorrhage following an operation on the tonsils, I have been profoundly alarmed at several of my own cases, and have seen some that resisted the skill and dexterity of experienced operators. The sinister and disconcerting fact is, in my mind, that patients have died from this cause. A colleague told me of a fatal case he had had in which the patient, a young girl of about fifteen years of age, swallowed quietly the steady ooze from the tonsillar cavity till she was exsanguinated. Her pallor finally attracted the attention of a nurse, who gave the alarm, but it was too late. This case had had nothing approximating a hemorrhage at operation, and there was no reason to fear any complication, though she really died of neglect on the part of the ward nurse, who should have watched her carefully.

E. A. Crockett, in stating that he considers hemorrhage a serious matter, says that in a year and a half twelve deaths from this have been reported in and about Boston, leading him to infer that there are many more than that number. However, in his discussion he brings out the fact that only one in every group of twelve untoward sequelæ of the tonsil operation was noted as occurring at the hands of a competent operator, meaning either a laryngologist or an operator who had had experience and training in this work. In addition to the twelve deaths referred to, he records "many serious hemorrhages," one requiring ligature of the common carotid after a scissors enucleation, and two deaths in two minutes, following a sharp dissection with the scalpel; truly an astounding calamity, absolutely inexcusable. None of these cases were his own, though he lost one patient from ether pneumonia followed by empyema, acute mastoiditis and meningitis.

Lindley Sewell was able to find reports of nineteen deaths, and fifty severe hemorrhages which were controlled. It is his opinion that the menstrual period conduces to greater loss of blood, if the patient be operated upon at that time. I have not been able to observe any difference, though I have repeatedly operated at this period and made careful notes on every case, in order to determine this point.

Ballenger, in nine thousand cases, had only two hemorrhages severe enough to cause marked anemia and exhaustion. Pfingst observed a hematoma in the pharynx as a result of suturing the pillars over a pledget of gauze to control a hemorrhage which began eight hours after operation. Cassellberry thinks that about one case in every seventy-five caused him some difficulty in stopping the flow of blood, out of an experience represented by fifteen hundred cases.

The case which caused me much anxiety was the result of a punch operation, coming on three hours after he had left my office. The flow was controlled with lumps of ice, fitted snugly into the bleeding cavity. Ten days later, while walking slowly on the street, his throat began bleeding and did not stop till he had fallen from syncope. I have seen twice in consultation an apparently uncontrollable primary hemorrhage due to failure to clean out thoroughly the supratonsillar fossa. A hard, fibrous nodule containing bleeding vessels was in each case discovered, and its snaring immediately stopped the flow of blood. I have observed that there are three points whence stubborn bleeding may arise in the radical operation: the supratonsillar fossa, the middle third of the posterior pillar, and the point where the infratonsillar fossa blends with the pharyngeal wall. In the method which involves divulsion of the loosened and hanging tonsil, there is apt to be a stripping of the small vessels in their length, which will always cause a type of bleeding necessarily stubborn, for obvious mechanical reasons. I have had three cases which required suturing of the pillars to control secondary hemorrhage, and innumerable ones in which I deemed it wiser to crush bleeding points or areas with powerful forceps rather than try pressure, though the latter method would have in time controlled the trouble, I feel sure.

Stucky reports a death from exsanguination a few hours after operation, and nine cases of secondary hemorrhage which required a second anesthesia and ligation or suturing the pillars. He does not give the names of the operators, or any particulars of the methods employed at operation.

C. W. Richardson, in a very frank and comprehensive paper on complications of the tonsil operations, reports no serious bleeding in his own cases, which he attributes to his technic of finger enucleation. In this I think he is correct, though I do only a partial finger separation and finish with the snare.

HYPERPYREXIA.

Richardson and Wishart both report a fatal case with this complication, in which the temperature rose to 107° F. The former case had a temperature of 99.4° F. at operation.

To be classed under this head, perhaps, is the case of a chauffeur, of good habits, whose tonsils I removed under the severe handicap of a badly given anesthetic. He was never sufficiently relaxed to dispense with the orderly's help, and the hemorrhage, from two torn vessels on the posterior pillar, rendered my task particularly difficult throughout. From normal at operation his temperature in two hours rose to 102° F. and in four hours to 105° F., and he was in a mild delirium for twenty-four hours. His chest, urine, blood and history were all negative. By lysis, in five days his temperature became normal, but it was a month before he regained his strength, though an unusually muscular man. All temperature readings were by axilla, which would show more fever than indicated by the figures 105° F., of course.

INFARCT OF THE LUNG.

Richardson reports two cases of this rare complication, in one of which it subsequently became necessary to open and drain a lung abscess. He also describes a case reported by Le Play, where the resulting abscess was probably due to an embolism.

SEPSIS.

It is probable that every case of tonsillectomy is complicated by some local infection, which is a natural consequence of laying bare two large granulating surfaces, with some trauma, in the presence of numerous bacteria. Certainly the three cases of cellulitis, one with the further issue of facial paralysis of five weeks' duration, reported by J. A. Stucky, would suggest this contingency. I have had five cases where swelling of the glands in the parotid region was sufficiently marked to require attention, though none went to suppuration. The presence of edema, which I have observed in at least 50 per cent of my cases, and have heard colleagues mention as a frequent complication in their work, however, is due to infection only in part, I believe, and has never caused any serious trouble in the convalescence. The removal of the tonsil in toto is likewise the removal of the support of the pillars and velum; hence, the relaxation and venous stasis contribute to the edema, if they do not actually cause it. The flabby, immobile palate allows food and liquids to pass into the posterior nares, simulating paresis; in fact, Sewell reports two cases of paresis of the soft palate which suggest to me merely a severely relaxed, edematous palate, and a patient naturally unwilling to incur great distress by voluntarily putting on the stretch such sensitive parts. Mild general sepsis is fairly common, accompanied by a temperature of 100° F., but severe cases of systemic infection, involving gangrene of cervical muscles, or septic phlebitis with optic neuritis and delirium, such as reported in Richardson's paper, as seen by L. W. Dean, are fortunately rare. Thus, Ballenger describes but two cases of his own, both due to the streptococcus. L. C. Deane says about 2 per cent of his cases showed this effect. Bauchacourt, in Dean's paper, reports one case of edema of the glottis with death, and Martin two deaths, while Dean gives one death from septicemia in his own practice. This occurred on the sixth day.

EMPHYSEMA.

Richardson's case, in which the air entered the subcutaneous area within an hour and cleared up in forty-eight hours, and the case of B. D. Parrish, where the emphysematous infiltration began a few minutes after operation, and extended from the right eyelid down the cheek, face and chest to the last rib, are the only cases I find recorded. I am indebted to Dr. Richardson's paper for these.

PNEUMONIA AND PLEURISY.

Scattered through the literature there is to be found an occasional report of pneumonia following a tonsillectomy, but as it is not always clear to the reader whether the death was due to the ether or to inspired matter, it is not easy to classify them. However, Richardson reports one pneumonia which proved fatal, and two pleurisies, neither of which was fatal. According to Crockett, there have been in recent years but two ether pneumonias at the Massachusetts Charitable Eye and Ear Infirmary, one of which died, but he has collected reports that show in the Boston neighborhood one death, which he says was due to strangulation from vomitus, and one death he calls an anesthetic death, though suspecting an enlarged thymus. A death from such a cause can in no way be attributed to the operation of tonsillectomy per se, but it is reported in this connection by the following, each of whom had one fatality: Dench, T. J. Harris, F. R. Packard. I have had one case of pneumonia which was certainly due to the manner of administration of the ether. His convalescence was very slow, and his temperature lasted for three weeks, but he suffered no permanent detriment.

RHEUMATISM.

Three days after removing the tonsils of a young boy, a slight swelling was observed on the outer aspect of each ankle, and a temperature of 100° F. This subsequently rose to 101.2° F., and the case ran a typical course and history of acute articular rheumatism, with involvement of the knees as well as ankles, lasting three weeks. I afterwards learned that he had been subject to such attacks every winter and in cold weather at other seasons. Apparently the operative interference had released latent infection in the tonsillar crypts and adjoining lymphatics with metastatic evidences in the joints mentioned.

NERVOUS PHENOMENA.

Pfingst mentions a case of hemiplegia which did not disappear till four months after operation, also one in which there were repeated attacks of hysterical strangulation and spasmodic flexing of the thighs (Bergh). Several months ago I saw in consultation an infant, fifteen months of age, from whose epipharynx lymphoid tissue had been curetted under chloroform anesthesia, for the relief of mouth breathing, the mother said. As an undoubted result of this unnecessary surgery, every afternoon since the operation this child had had violent attacks of spasmodic laryngitis, with complete arrest of respiration for as long as fifteen and even twenty seconds at a time, and all the cyanotic and cardiac disturbance which usually accompanies such seizures. The child had a negative history, had neved been ill and had parents of no taint whatever. Evidently the violence done her nervous equilibrium was great and causative of the attacks.

The rather severe edema of the uvula and soft palate following an enucleation of the tonsils in a neurotic woman, twenty-five years of age, had begun to disappear when I was sent for in great haste on the fifth day, as she was unable to speak, even in a whisper. The cords appeared normal and moved freely on attempted phonation, and no cause other than hysteria seemed to me likely at the bottom of the aphonia. I tried to betray her into speaking by catching her off her guard, but knowing that she was very desirous to go to Europe in a few days, I told her mother in her presence that her voice would return unimpaired by the second day at sea. This was sufficient to secure a cure, as promptly the second day, on getting up, her voice was found to be full and normal.

INJURY TO PILLARS AND ADNEXA; CONTRACTURES; FUNCTIONAL DISTURBANCES.

Laceration of the pillars is a frequent occurrence where they are friable from disease, or where much force, or rather misdirected force is applied, and will materially increase hemorrhage, especially when the posterior pillar is the part damaged. Generally these wounds heal readily with no ill effects other than prolonged convalescence, but I had the misfortune several years ago to buttonhole an anterior pillar with the punch, and the wound did not close. Moreover, the cicatricial contractures were extensive, and the two pillars adhered together on each side, limiting the mobility of the velum in such a way as to render the patient's voice nasal from time to time, though not continuously. Since abandoning the use of the punch some three years ago I have had no such perforations or contractures. While I do not seek to escape the blame for my poor technic that brought about such a result, I find myself in accord with Sewell, who says that many of these bad cosmetic and functional results are due to the faulty administration of the anesthetic. It is essential for good results in throat surgery that the patient should be kept quiet, the mouth free of blood and mucus and the anesthetic apparatus out of the operator's way. My case bears out his theory, as the three conditions desired were conspicuously lacking.

I was rather surprised to read that Pfingst found it not unusual to buttonhole the pillars in the first twenty-five or thirty cases he had to report, and the uvula was at times removed along with the tonsil. This I have done once with the snare, and I have seen two eminent laryngologists do the same thing in this city. If the snare loop is made so small that the loosened tonsil can be just pulled through with slight force, it will be impossible to commit this surgical error, which I have likewise done once with the punch, as the parts were obscured with blood and the patient not well anesthetized. In all, I have had four cases in which the scar formation was marked, all due to indiscreet use of the tonsil punch.

Sewell reports one case of adhesion of the velum to the pharyngeal wall, shutting off one naris from the oral cavity. I had a similar case in which the velum was adherent to the pharynx on both sides, leaving an opening in the midline which scarcely admitted my little finger. As he was a mental defective, an accurate history was not obtainable, other than it was the result of a throat operation.

Where a tonsil is badly degenerated and the pillars share in the process, though the tonsil be removed with the utmost care and circumspection, the rapid formation of granulations requires removal for weeks following convalescence, if deformities are to be prevented. Such has been my experience with tonsils, the seat of infection spreading into peritonsillar abscess, and requiring removal as a prevention of recurrent attacks, though I try to prevent this new growth by local applications to the healing surface. One case, that of a woman of forty-five years of age, required free punching out of large organized granulations three times in the course of three months.

The examination of a young man's throat for the relief of pain and offensive accumulations revealed the absence of a uvula and the presence of tonsil remnants on each side, though he had had a general practitioner remove the tonsils once, and again a specialist had tried to do it, with the result already stated. Moreover, the free edges were the seat of chronic inflammation, their surfaces ulcerated and covered with a pinkish and yellow material, and a glistening, tense white band of fibrous tissue bounded the free edge of the pillars on one side, up and across the palate and down the edge of the opposite pillars, holding the velum in a tight grip. As the patient was pale, weak and dispirited, I would give no prognosis till I had a report from the pathologist who examined a section from each tonsil, with a negative result. As there was no glandular involvement or temperature, the tonsil base on each side was enucleated, revealing a sclerotic mass the size of an English walnut—true tonsil tissue. His voice is occasionally nasal, though the contracture is better than before my operation.

A clean removal of diseased or enlarged tonsils will at times increase the register of a singer's voice as much as two tones, but, on the other hand, it is unwise in the extreme to operate on the throat of a singer without first warning the patient what may be the outcome as to limitation of tone production. However carefully the removal be done, there will sometimes be a restraint placed on the palatal musculature by scar formation and adhesion of parts formerly free, such as to impair tone production and resonance. Such cases are on record; in fact, Stucky refers to one in his report.

Hudson Makuen goes into this particular point thoroughly and vigorously, laying stress on the carelessness with which the soft parts are handled. His explanation of why adhesions and fibrous formations alter the voice is particularly lucid, and I speak only in corroboration, since nothing can be added to his work save further evidence.

Whether we regard the enucleation of tonsils as an operation free from peril, or distinguished by an occasional mishap, at least the conviction forces itself upon us that it should not be entrusted to any but those experienced in its performance, and that the convalescence should be zealously followed. Moreover, it is essentially a hospital operation, to be done in a hospital or under the same rigid condition of asepsis and trained nursing.

BIBLIOGRAPHY.

1. **Richardson, C. W.:** Trans. American Laryngological Assoc., 1912, pp. 170-183.
2. **Freer, O. T.:** Idem., p. 184.
3. **Sewell, Lindley:** Medical Chronicle, Manchester, July, 1911, p. 212.
4. **Wishart, J. B. G.:** Laryngoscope, February, 1909.
5. **Le Play, A.:** Archives Genéral. de Méd., 3280, 1905.
6. **Dean, L. W.:** Laryngoscope, Vol. xx, No. 7, p. 738.

7. Ballenger, W. L.: Diseases of Nose, Throat and Ear, 1911, Ed. 3, pp. 416, 428.

8. Packard, F. R.: American Jour. Med. Sciences, September, 1910.

9. Deane, L. C.: Cal. State Jour. Med., 1909, p. 92.

10. Harris, T. J.: Annals of Otol., Rhinol. and Laryngol., 1909.

11. Crockett, E. A.: Boston Med. and Surg. Jour., March 23, 1911, p. 414.

12. Bergh, E.: Monatschrift für Ohrenheilkunde, Berlin, 1904, Vol. xxxviii, p. 549.

13. Pfingst, A. O.: Laryngoscope, July, 1911, p. 798.

14. Makuen, G. H.: Trans. American Laryngological Assoc., 1911, pp. 223-236.

15. Cassellberry, W. E.: See article of A. O. Pfingst (13), p. 804.

16. Dench, E. B.: Idem. (13), p. 804.

17. Stucky, J. A.: Laryngoscope, October, 1912.

18. Parish, B. D.: Laryngoscope, November, 1910.

19. Bauchacourt: See Dean's paper (6), p. 741.

20. Martin: Idem., p. 741.

LXIII.

DECAPITATION OF THE FAUCIAL TONSIL FOLLOWED BY DIGITAL EVISCERATION OF ITS CAPSULE.*

By J. A. Ellegood, M. D.,

Wilmington.

For nearly three years it has been the almost invariable practice of the writer to eviscerate the tonsil capsule, after decapitation, when operating for the removal of the faucial tonsils in children under puberty. In nearly all of the cases the evisceration was performed by means of the index finger.

The patient is prepared as for a tonsillectomy. A general anesthetic is administered. A digital examination of the tonsils is made to determine the consistence of the tissues of which they are composed. If sufficiently soft, as it is in most cases, the hypertrophied mass is pressed through the ring of a Mackenzie tonsillotome of suitable size and the decapitation performed. In the pedunculated tonsils preliminary dissection is often unnecessary. In the case of imbedded tonsils it is generally necessary to separate the tonsil from its attachment to the faucial pillars before it can be made to protrude through the fenestra of the guillotine. The separation can frequently be performed by the index finger, the tip of which can usually be pressed up under the margo supratonsillaris into the supratonsillar fossa and brought down successively between the anterior and posterior pillars and the tonsil. During this procedure much assistance can be rendered by supporting the region of operation externally by the hand of the operator or an assistant.

In case the separation cannot be satisfactorily done by the finger, the tonsil may be grasped with forceps and instrumental means made use of for its accomplishment.

---

*Candidate's thesis, American Laryngological, Rhinological and Otological Society, May 9, 1913.

In the ideal performance of the decapitation the incision should be at the junction of, or slightly inward to, the point at which the capsule and the mucous membrane meet as the later is reflected over the tonsil. Such an incision avoids cutting the strong fibrous membrane, a part of the amygdalo-epiglottic fold, which lines the whole tonsil fossa and lies in front of the loose cellular tissue between it and the superior constrictor muscle.

The extent to which the tonsil extends through the ring of the guillotine can, in a great measure, be determined by the pressure of the surgeon's finger externally, at a point corresponding to the interval between the angle of the lower jaw, the anterior border of the sternomastoid muscle, and the great cornu of the hyoid bone.

Should the pressure be made by an assistant and be excessive, the degree of protrusion can be regulated by the operator in applying the instrument.

In a small proportion of cases of pedunculated tonsil the entire tonsil in its capsule will be removed by the guillotine. In the majority of cases, however, the base of the tonsil and its capsular bed will be left in situ. The index finger, armed with a stout nail, previously hardened with absolute alcohol, is introduced and the remains of the tonsil tissue broken down and scraped out. This is usually easily accomplished. Ordinarily no great amount of force is necessary to so crush or pulpify the tonsil tissue that it may be easily curetted from the capsule by the finger nail. Any that may remain attached to the edges of the capsule can readily be removed by tongue or other suitable forceps, or by a small Ballenger ecraseur.

The crushing of the tonsil tissue may be aided by external counterpressure, or the mass in the capsule can be easily pulled forward by the finger over the angle of the lower jaw, which can be used as a background.

An examination of the decapitated part will enable the operator to determine the consistence of the remaining stump. Should that, as occasionally happens, be too tough for complete removal by digital manipulation, it can be removed by such other means as the operator may elect to adopt. As a matter of experiment the lymphoid tissue in stumps of tough consistence has been removed, leaving a considerable portion of the fibrous tissue. In all such cases the remaining tissue soon atrophied, demonstrating that the presence of lymphoid tissue is in most cases necessary to excite hypertrophy of the fibrous elements.

Not infrequently the lymphoid tissue is divided into two or three distinct masses or lobes by more pronounced septa of connective tissue which extend from the inner surface of the capsule. These should be broken down and the cavities converted into one.

It is important to remove all the lymphoid tissue. Shreds of connective tissue and the ends of the vessels which remain, speedily disappear.

If in making the section the incision does not involve the greatest diameter of the investing membrane, it should be torn or cut at its lowest part to permit of easy drainage. It can usually be easily torn with the finger.

The crypts are doubtless the cardinal features, both as regards the function and the pathology of the faucial tonsil. Of the former nothing definite is known. Normally the tonsils disappear after puberty. Although they may have some useful function to perform before this period, it has been unquestionably demonstrated that their absence by complete removal in no appreciable sense prejudices or endangers life or health. As yet no bodies have been discovered which bear the same relationship to the tonsil that the parathyroids do to the thyroid gland. It is possible that such may exist, or that some of the other lymphoid tissue constituting the "ring of Waldeyer" may vicariously, or by substitution, perform whatever part the tonsil is intended to accomplish in the process of metabolism. The crypts of largest size and greatest depth are, as a rule, found in the middle part of the tonsil, and this point is the place most desirable for beginning the process of digital evisceration.

While some of the crypts open into the supratonsillar space, none exist in any of the lymphoid tissue composing "Waldeyer's ring" outside the tonsil capsule.

The advantages claimed for the operation herein described are that in most cases it is easily performed. It is also quickly performed, thus lessening the danger of profound and prolonged anesthesia. Less assistance is required, as mopping of blood and mucus is unnecessary. The patient's head can be held in such position as to prevent the inspiration of either of these substances. No inspection being necessary, they do not interfere with the performance of the operation. The danger from bleeding is lessened. There is no injury to the faucial pillars, nor to the venous plexus at the base of the sinus, the wounding of which is the most common cause of persistent and alarming hemorrhage. It is obvious that the more peripheral the incision the smaller the vessels cut, and the less serious the hemorrhage.

The vessels and fibrous septa are too tough and resistant to be entirely removed by so dull a curette, and, stripped of lymphoid tissue, the former readily retract and curl upon themselves so as to close their lumen. The danger from sepsis is less than when the capsule is completely removed, because the wound is less extensive and deep, and has as its background a tough and resistant fibrous membrane, instead of loose cellular and muscular tissue. In a number of instances in which the tonsils filled the entire fossa they were so covered by the plicæ that there appeared to be no peritonsillar space, and there being little or no projection beyond the faucial pillars, a section of the mucous membrane was removed from the buccal surface of the gland and evisceration digitally performed. The incubating foci represented by the crypts being eradicated, and the mucous membrane and capsule being torn below so as to afford drainage and avoid the formation of a pocket, the result was all that could be desired.

The upper portion of the capsule is surrounded by a loose areolar tissue space—the peritonsillar space. There is only a potential space round the lower portion of the capsule, for the muscular fibers of the palatoglossus and the palatopharyngeus form a close investment to it. These fibers are interlaced with one another and with those of the superior constrictor, and some of the latter are directly inserted into the capsule itself. Consequently this intimate relationship of muscle with the lower part of the capsule prevents tracking downward of infection when the lower part of the capsule and its attachment are not disturbed, whereas, such might easily occur when the lower part of the capsule is removed. The great resistance of the capsule to infection is well illustrated in gross tubercular lesions of the tonsil. In tonsils which have undergone acute inflammatory changes the capsule remains unaffected. So it apparently does after digital evisceration. Lastly, there is no mutilation of the palate muscles, nor such injury to the mucous membrane as to interfere with deglutition, audition, or vocalization.

It may be said that such injuries are not done when total extirpation of the capsule is properly performed. While that is undoubtedly true, nevertheless, it is a well known fact that they are frequently done by the inexperienced, and occasionally by the most expert operators. It may also be said that the force necessary to perform the operation described is liable to cause serious injury to the surrounding parts, and that the remaining shreds of tissue in the eviscerated capsule are liable to lead to general infection. In the experience of the writer there has been, as a rule, but moderate reaction, the detritus has rapidly disappeared, and the denuded surface was speedily covered by extension of the mucous membrane. The insult to the tissues is by no means so great as in the old operation of Celsus—digital enucleation—revived and practiced by some distinguished surgeons of the present day.

While in some instances the child is born with hypertrophy of the faucial tonsils, the enlargement usually begins about the second year of life, and continues until young adulthood. During this age the lymphoid elements largely predominate, and the consistence of the tonsil is such that digital evisceration can be easily accomplished in the manner described. Although this operation may not commend itself to skillful surgeons, the author is convinced that it is based on good surgical principles. The results in over three hundred cases performed by him have been eminently satisfactory. In only one case has there been any recurrence of the hypertrophy, and in that case circumstances arose during the operation which prevented its complete performance. The hypertrophied mass in this case is quite smooth, by no means large, and has given rise to no disturbance. There has been no case of alarming hemorrhage, and in but one was there violent reaction. That one was operated upon too soon after an acute attack of tonsillitis, and although there was considerable reaction, recovery was complete and the final result perfect.

Simple tonsillotomy in most cases is unsatisfactory, because it leaves portions of the crypts, some of which almost invariably extend through the entire depth of the tonsil to the capsule, and these are liable to become subsequently diseased. Nevertheless, it is a time-honored procedure and, except in America, the method of operation most commonly performed. Unless some operation more efficient and more easily executed is devised, it is a procedure likely to be largely performed in the future. Tonsillectomy, as performed by the most expert operators, is a more difficult operation than the average operator will practice, and complete removal by means of a tonsillotome and punch forceps, with or without removal of the capsule, is both more difficult and dangerous than tonsillectomy.

LXIV.

FRONTAL MUCOCELE.*

By Geo. W. McCoy, A. M., M. D.,

Los Angeles.

The profession differs in the definition of a mucocele, the difference being essentially in the location of the tumor. Defined in the words of St. Clair Thomson, and representing the views of the majority of nose and throat men, a mucocele is a "distension of one or more walls of an accessory sinus and an accumulation within it of a mucous secretion."

This conception of a mucocele restricts the growth to an uncommon occurrence. Hill Hastings, in his excellent article read before the society at Los Angeles, 1911, reviewing the literature, found only forty cases.

The other conception defines the term, in the language of D. Braden Kyle, as a "simple or retention cyst common in the nose and nasopharynx and the upper portion of the larynx." He also describes the condition in the accessory sinuses.

The etiology is uncertain. Turner believes a catarrhal process obstructs the natural ostium and the mucous secretions are retained, but there are cases in which the passage is patulous. The usual cause of mucocele, according to D. Braden Kyle, Thomson, and others, is cystic dilatation of a gland of the mucous membrane, or cystic degeneration of a polyp, but may be due to catarrhal stoppage of the ostium. This is called by the recent writers the new belief, and the obstructing cause is the old idea, yet some recent writers, accepting the new etiology, still restrict mucoceles to the accessory sinuses.

As there is a greater number of myxomata and fibromyxomata in the nose than in the accessory sinuses, and as the mucous membrane of the nose and nasopharynx contains more glands and has a greater number of cyst formations than the mucous membrane of the accessory sinuses, it is evident that conditions are far more favorable for the development of mucoceles in the nose and nasopharynx than in the sinuses. In my own experience I have removed a few tumors from the nose and nasopharynx which, if placed in the accessory sinuses, would be considered by all mucoceles, while I have only had two cases of mucocele of the accessory sinuses, and possibly a third, now under consideration.

---

*Read before the Western Section of the American Laryngological, Rhinological and Otological Society, Oakland, April 15, 1913.

Restricting the term mucocele to such conditions within an accessory sinus is artificial, and is not founded upon pathologic principles, and should be discontinued. The term should include any simple or retention cyst filled with mucous secretion which may become purulent or undergo secondary changes.

The following case would be a mucocele under either definition: A young man, thirty-four years of age, weighing 204 pounds, five feet nine inches in height, and in the pink of physical condition, a baseball catcher by profession, was referred to me by Dr. Guy Cochran for a tumor over the left frontal region as large as an English walnut.

The family and personal history, except the following, has no bearing. Denies venereal and alcoholic history, excepting an occasional glass of beer. Had socalled typhoid malaria at fifteen years of age; had measles when a child; with the exception of an occasional attack of grippe, has otherwise been in perfect health.

The patient has had difficulty in breathing through his nose the mucous membrane, or cystic degeneration of a polyp. but the patient's story, Dr. Rae of Binghamton, New York, removed some cartilage and bone from his nose, without much relief. The patient was advised to go west and get into the open air. He came to California in 1904 and improved immediately, probably as the result of Dr. Rae's operation. He caught ball for the Seattle and Sacramento teams, and then went to Salt Lake City, having little or no catarrhal trouble until 1910, when headaches and a full feeling appeared over both eyes, not following or accompanied by the slightest cold nor stoppage of the nose. Under treatment by Dr. Pfouts the trouble disappeared for six or eight weeks. In January, 1911, headaches and the full pressure feeling over the left eye returned. Soon a swelling appeared over the inner third of left frontal sinus region, about the size of end of little finger. When the tumor appeared headaches ceased, but pressure feeling was always present. Dr. Landenberger aspirated a dark greenish fluid and a small opening in the frontal sinus wall was noted. The pressure feeling left, but in a month returned with the reappearance of the swelling. He was again aspirated; the patient was then sent to Dr. Schramm, who operated from within the nose, removing the left middle turbinated body. According to the patient, three weeks after the operation the contents discharged through the nose. The tumor mass soon reappeared and again discharged itself and again reappeared.

In the spring of 1912 I found a nonfluctuating tumor of the size of an English walnut over the left frontal region. The skin over the swelling was normal in appearance, but intimately connected with the mass. No opening through the bone could be felt. The left middle turbinated body and the anterior ethmoidal cells had been removed. The right middle turbinated body was hypertrophied. There was a slight mucous discharge from the nose, but none from the sinuses. Transillumination was dark over the left frontal. Dr. Soilands' X-ray plates showed the extent of the sinus and as if slightly blurred, also the tumor mass over the anterior wall of the frontal sinus, but did not show the opening in the bone. I observed the patient until August, 1912, and at no time found any discharge from the frontal sinus region. I aspirated about an ounce of a dark brown, slightly thick, but not tenacious fluid, sterile on agar and blood serum. The opening in the anterior frontal wall could be distinctly felt. Repeated and careful examination of the eyes, ears, nose and throat revealed no symptoms except those given August 31, 1912. I operated at the Good Samaritan Hospital. An incision was made along the brow and down the side of the nose. The periosteum and the soft parts were carefully elevated and retracted. An opening at inner third of the anterior frontal sinus wall, irregular in shape, about 8 mm. in diameter, was found. A small sequestrum was within the cavity, excessive bleeding clouded the liquid contents. The bony interior of the sinus throughout looked like the smooth whitish interior of a muscle shell without any membranous cover, nor could the sac of the mococele be made out. The lining of the retention sac of the soft part had this smooth appearance, but not so white in color, nor could a distinct sac of the mucocele be made out here. No dura was exposed. The nasal ostium was completely closed, and there was not the slightest sign of the sinus ever having had an outlet—just a small pit covered with pearly white bony lining where the passage should be. Through this I made a nasofrontal passage with difficulty, coming down by mistake upon the septum so that I had an opening into either nasal cavity. The anterior bony wall of the sinus being thin was entirely removed, the cavity packed with iodoform gauze and the wound entirely closed with horse hair sutures. The gauze was removed next day from the right side of the nose.

The recovery was uneventful, healing by first intention, and on the tenth day the patient left the hospital. There was slight diplopia for a month. Six months later I showed the patient to the Los Angeles eye, ear, nose and throat section. There is no deformity, no depression. The scar, owing to the slanting incision through the skin, cannot be seen over the brow, and down the side of the nose only as a faint line. At first I could pass a probe from either nostril up into the cavity. Ten days ago I attempted it and did not succeed.

Dr. Lund, who was present, advised letting the external wound heal by packing from without, avoiding making a new passage to the nose, not disturbing the mucous membrane of the nose and lessening the danger of discharge from sinus into nose. To me it seemed more or less deformity would result; the interior of the sinus I feared was too smooth to granulate, and as aspiration had been done three times, I thought the nasal drainage method the best. The patient is well. I cannot reconcile his statement of the tumor mass emptying itself twice through the nose, unless the passage was later closed.

LXV.

REPORT OF A CASE OF COMPOUND FOLLICULAR ODONTOMA OF THE SUPERIOR MAXILLA.*

By L. W. Dean, M. D.,

Iowa City.

Miss C., age twenty years, presented herself for examination on March 6, 1912. The following history was given me by Dr. Bowie, who referred the patient to me. He reported that the patient presented herself to him several weeks before, complaining of pain in the region of the upper right third molar. The third molar was sensitive. Treatment of the tooth with removal of the nerves from the root canals did not relieve the pain, and two or three weeks before I saw the patient the tooth was extracted. The tooth had the usual short. conical root. It was two weeks after the tooth was extracted that a discharge of seropurulent matter made its appearance in the socket of the tooth, and a number of rudimentary teeth were discharged through this opening.

Examination showed that there was a sinus leading upward from the root socket of the right third upper molar. On probing this sinus it was found to open into a large cavity. The diagnosis of odontoma was made and operation advised.

The opening was made into the superior maxilla, just above the gingival border, over the socket occupied by the third molar tooth. A large cavity was found which extended forwards over and external to the first and second molars. The cyst extended into the hard palate one-half the distance to the median suture. Within the cavity were found a number of rudimentary teeth and irregular pieces of bone tissue. In all, preceding the operation and at time of operation, thirty-five pieces were removed. The antrum was opened and found normal. The wound was drained through the antrum into the nose and the external opening closed. Later the wound reopened and was closed by a second operation.

---

*Read before the Chicago Laryngological and Otological Society, April 22, 1913.

In connection with dentigerous cysts and empyemas of the antrum of Highmore, with extensive necrosis and the operation for these conditions, I have been experimenting with various methods for closing the wound between the cavities and the mouth. The method that I have found most successful was to remove the bone from the alveolar process so as to get good apposition of the periosteum covering the external and internal surfaces of this process.

I found that if the wound were closed with silk sutures they would cut through and the wound reopen. Catgut was next tried with similar result. Our best results have been secured by using silkworm gut and placing along each side of the alveolar process a small piece of very narrow rubber tubing. Over the tubing, on the inner surface of the process, the silkworm gut is looped, the ends being tied over the tubing on the external side of the process. In all, during the last eight months, I have operated on some fifteen cases. The last seven or eight have been operated upon by this method with uniform good results.

It might not be amiss here to quote from the most recent authorities in regard to odontomata. It is evident that but little is positively known except as to clinical experience and treatment. The etiology is still within the realm of speculation. There are several kinds of odontomata, or tumors composed of one or more of the dental tissues arising either from teeth changes, or teeth in process of development.

Sutton classifies the different forms of odontomata as follows:

Epithelial Odontomata.—These develop from the remains of the epithelium of the original enamel organ. They appear in the form of a series of cysts separated by thin septa and contain mucoid fluid.

Follicular Odontomata or Dentigerous Cysts.—They usually form in connection with developing permanent teeth. The inner wall of the cyst is the remains of the dental follicle, within which, in every instance, there is a tooth or remains of a tooth. The cyst is filled with fluid, usually serous or mucoid.

I believe this fluid always contains cholesterin crystals. The contents are not purulent unless infection comes from without. The walls of the cyst are bony. They may become so large as to bulge out and cause great deformity.

Fibrous odontomata are developed from the connective tissue elements of the developing teeth. These are usually seen in animals.

Cementomata, the result of cement forming around the teeth, are rare in man.

Radicular odontomata form after development of crown and while root is forming.

Composite Odontomata.—These are abnormal growths of all the elements of tooth structure, resulting in solid, illformed masses of cementum, dentine, and enamel.

Compound Follicular Odontomata.—Socalled because it results from several tooth follicles. The follicle wall becomes thickened into a fibrous capsule. In this there may be portions of dentine, enamel, or cementum, imperfectly or more or less perfectly formed teeth, all together in a heterogeneous mass. Rudimentary or other teeth have sometimes been found in very large numbers; as many as several hundred have been taken from the jaws of one person. (Oral Diseases and Malformations, Brown.)

These tumors have been observed in the human subject, and in goats, horses, and other animals. In man they usually develop in early life, during the period of second dentition. Among the records of cases of this character the youngest was a boy ten years old, and the eldest a woman of twenty-seven years.

The number of denticles found in tumors of this character varies greatly. From three to four to as many hundreds have been removed from the jaws of a single patient.

Among the recorded cases of especial interest is one which will be briefly mentioned:

Tellander reported a case of tumor of the right side of the superior maxilla in a woman twenty-seven years of age, which had been growing since she was twelve years old. The character of this growth was a hard, painless swelling. It occupied the space in which should have been lodged the first molar, the bicuspids and the cuspid of the permanent set. These teeth had not erupted. Upon opening the tumor it was found to contain several minute teeth; nine individual teeth with conical crowns and conical roots, each complete in itself, the crown being furnished with enamel, and six masses of dental tissue having the appearance of being formed by a union of several single teeth, and all presenting the characteristics of supernumerary teeth. Another tooth made its appearance about a year later in the location from which the tumor was removed. (Ottolengui, Items of Interest, February, 1913.)

LXVI.

CASE OF MENINGITIS IN WHICH THE CISTERNA MAGNA WAS DRAINED.*

By Norval H. Pierce, M. D.,

Chicago.

O. L., aged ten years, schoolboy.

Diagnosis.—Mastoiditis; purulent meningitis.

History.—Patient had had a chronic suppurative otitis media for nearly two years.

Present Condition.—Temperature on admission was 101.6.° Some rigidity of neck. Kernig present. Babinski. Pupils not contracted and eye grounds apparently normal, excepting that the veins appeared just a trifle large.

Patient was admitted to the hospital on March 10, 1913. The following day the leucocyte count was 7,500. Lumbar puncture was made, and fluid came out with considerable pressure and was cloudy. On March 12th, examination by Dr. Pierce showed spontaneous nystagmus on both sides. Heard a^1 fork. No symptoms of fistula. Radical mastoid operation was performed by Dr. Pierce on that day, and necrosed bone found in roof of tympanum and posterior wall of mastoid, extending back along the skull. Black necrosed bone removed and dura exposed. The next day the patient was no better—all the symptoms of meningitis were the same. Lumbar puncture: ten cubic centimeters, under pressure, cloudy. Specimen sent to Michael Reese Hospital, where cultures were made and pneumococcus in pure culture reported present; one hundred and sixty cells to field, polymorphonuclear predominating; Noguchi plus.

March 14th.—Lumbar puncture—twenty cubic centimeters. Marked pressure; cloudy. Stiffness of neck more marked. Kernig marked. Babinski. Patient cried out constantly and complained of severe headache.

---

*Read before the Chicago Laryngological and Otological Society, April 22, 1913.

March 15th.—Drainage of cisterna magna by Dr. Pierce. A trephine opening was made one inch below external occipital protuberance and the opening enlarged with rongeur forceps. No pulsation present. Dura and arachnoid opened and considerable cloudy fluid allowed to flow out, and immediately pulsation was seen. A gutta percha drain was inserted and two pieces of xeroform gauze inserted external to dura, one above and one below gutta percha drain, and the wound closed. Voluminous boric and alcohol dressing applied. Blood pressure taken during the operation fluctuated between 110 and 95.

March 16th.—Patient's temperature went down after the operation for several hours, but again up to 101° on evening of the 16th. Leucocyte count, 3,000.

March 17th.—Wound dressed twice a day. Drainage. Blood pressure went down to 75. Patient's limbs and abdomen bandaged and strychnin given. Blood pressure went up to 95.

March 18th.—Temperature, 101° to 103.° Wound dressed twice daily and draining slightly. Blood pressure 90. Patient cried out often and complained of some headache. Emaciation marked.

March 19th.—Temperature, 100° to 102.° Headache, and patient cried out. Drain out of wound. Wound reopened by Dr. Pierce, allowing considerable cloudy fluid to escape and rubber drain reinserted. Blood pressure down to 80.

March 20th.—After reinserting drain, temperature from normal to 99° for twelve hours, after which it again went to 101.°

March 21st.—Patient was getting stupid and showed evidences of marked sepsis. Temperature, 101° to 102.° Blood pressure 90. Involuntary stool and urination at times. Patient not unconscious, but slept a great deal, but perfectly rational when aroused. Wound was draining, but looked dull and septic.

March 22nd.—Patient much worse; sepsis more marked; marked spontaneous nystagmus; stuporous, but could be roused.

March 28th.—Patient died.

We have here a case of undoubted septic meningitis. All the classic symptoms are present—stiff neck, engorged veins in the retina, Babinski, Kernig, headache, spinal fluid showing a large percentage of white and polymorphonuclear corpuscles and a virile microorganism. It is interesting to note that the germ which was grown from a spinal puncture apparently differed from the germ which was procured directly from the cisterna magna. However, I believe that this is a bacteriologic error, as the germ which was taken from the cisterna magna resembled very closely but was not the pneumococcus, and differed from it principally in that it was hemolytic.

We depend for our diagnosis on lumbar puncture in meningeal inflammations. Still, the findings are not always useful in differentiating between serous meningitis, localized meningitis which is permanently circumscribed, diffuse meningitis, or one that is spreading. In all probability, if we can recover a live pathogenic bacterium from the spinal fluid, we have to do with meningitis which is already diffuse or is at the time spreading. Other than this, we may have an enormous cytologic content in either a serous meningitis or a meningitis which is permanently circumscribed.

It is very important, in contemplating the performance of this operation—draining the cisterna magna—to differentiate between a serous meningitis, a circumscribed meningitis, and a diffuse meningitis, for I do not believe that the operation is indicated in anything but a diffuse meningitis, or a meningitis which tends at the time to spread. The two former conditions —serous meningitis or circumscribed meningitis—may best be treated, I believe, by spinal puncture or permanent lumbar drainage. If the base of the skull is bathed in pus—in other words, if the cisterna is an abscess cavity—then this operation is indicated. I can readily imagine how the unavoidable trauma incident to the operation of draining the cisterna magna, the extreme and sudden depletion of the subarachnoid space of its natural fluid. might transform a circumscribed meningitis into a diffuse meningitis. The fact that it has the advantage over splitting the dura in the mastoid wound (a septic field) is to a certain extent true, for in our case the external incision healed by first intention, and when the drain had to be replaced it was found that the dura itself had largely healed; it cannot be considered as aseptic or as free from the danger of infection as, for instance. a surgical wound made in the skull in the course of an operation for tumor, for in our case the septic wound in the mastoid is only a short distance from the wound in the occiput, and both are covered practically by the same dressings.

How, then, will we decide as to when is the proper time for opening the cisterna magna—for this operation has a place in surgery. If the headache is severe, temperature high and stiff neck increasing, we should depend first on lumbar puncture. The cytologic, bacteriologic and chemical condition of the cerebrospinal fluid should be carefully studied. In the absence of living bacteria we should continue with our lumbar drainage. If, however, the cytologic content increases and the clinical picture is that of an increasing meningitis and we recover a living pathologic bacterium from the fluid, we may resort to opening the cisterna magna. However, I would raise my voice in warning against participating in a hope which, in my opinion, will be vain.

There are two factors with which we have to deal in a meningeal inflammation: First, and least important, the increase of the cerebrospinal fluid. Second, the inflammatory process which takes place in the pia. It is the latter process which kills, and we may drain off the cerebrospinal fluid for days, and even weeks—as we did in our case—and the patient succumb to the meningitis. It is the inflammation about the sheaths of the blood vessels which is carried in the pia to the vital center of the brain and medulla which kills. I doubt if subdural drainage of any kind will influence the disastrous process in the pia, once it has become diffuse.

The operation itself is easy of performance. The technic has been thoroughly described by Dr. Haines. I believe, however, it is right to caution against the too sudden release of the fluid, which is usually under great tension, from the cisterna magna. This may best be obviated by introducing a needle through the dura and arachnoid after the bone has been removed. It will take some time, of course, before sufficient fluid has been removed in this way, before the pressure has been reduced to any considerable extent, but there is no reason why a very small capillary tube should not be inserted into a very small opening at the first sitting and then, after another subsequent dressing—say within the next twenty-four or forty-eight hours—the dura can be fully incised, if necessary, and examination of the foramen of Magendie made.

The fall in the blood pressure is a very dangerous sign. It may go very low, however, without terminating fatally. We should be ready at the time of operation to bind the extremities and the abdomen in case of syncope, and afterwards, in the course of the disease, when the blood pressure gets below 100, the same procedure should be carried out.

LXVII.

THE USE OF VACCINES AFTER THE MASTOID OPERATION.

By William C. Braislin, M. D.,

Brooklyn.

I have employed vaccines in six cases, subsequent to the mastoid operation, out of a total of thirty-five mastoid operations performed during the past winter. In the ordinary, smoothly convalescing cases they have not been used. They were used in cases presenting the following complications:

1. Extensive infiltration of the glands of the neck following an operation for streptococcic mastoiditis and cervical abscess.
2. Indeterminate or obscure causes of elevated temperature (the lateral sinus excluded from involvement).
3. Delay in granulation and healing processes in mastoid wound.
4. Pain in limbs, joints and back, without special elevation of temperature, after an operation for streptococcic mastoiditis.
5. In a case of postoperative pneumonia.
6. In a case of infection of the labyrinth.

Case 1.—Streptococcic infection of right mastoid in a little girl of two and a half years. The mastoiditis was complicated in this case by a very extensive abscess on the corresponding side of the neck, which was operated upon at the time of the mastoid operation and a drainage tube introduced into the depth of the wound. The cervical abscess was deeply seated and surrounded by enlarged lymphatic glands, one of which protruded into the right lateral and posterior wall of the pharynx. A culture taken at this time and submitted to Dr. Archibald Murray showed the infection to be streptococcic. The child did well for three days after the operation, but on the following day the lymphatic glands of the neck on the opposite side became tense, markedly swollen and tender, especially at the angle of the jaw, and a distinct rise of temperature occurred. No involvement of the mastoid was discoverable on this (the left) side. The culture made at the time of the operation not being available for making an autogenous vaccine, we began the use of a stock (antistreptococcic) vaccine, a minim being given daily for four days, then on alternate days for four days more. The glandular swelling had begun to decline within twenty-four hours of beginning treatment. The discharge of pus from the wound lessened rapidly likewise. The temperature became gradually normal, and there appeared to be a sensible increase in the rapidity of the healing of the mastoid wound. The neck wound healed within three weeks from the time of operation; the mastoid wound, at the end of the sixth week.

Case *2*.—Pneumococcus infection of both mastoids, ten days after eruption of measles, in a girl three years old. Both drums were promptly incised. Cultures taken from both ears were reported by Dr. Murray to be pneumococcic. The discharges continued to be profuse from both ears, and the temperature continued high. The right mastoid, which was tender to pressure, was operated upon, the sinus being exposed but healthy. The antrum and cells contained much free pus. An autogenous vaccine from a culture removed from the antrum was started in course of preparation, at our request, by Dr. Murray. Much to my disappointment, the temperature did not subside to normal, but continued nearly as high as before the operation, between 102° and 104,° so that after excluding the lungs from any influence in the production of the temperature, operation was performed on the left mastoid on the third day following the operation on the right. Flaky pus was found in the antrum. We now believed that the temperature would fall to normal, but in this we were again disappointed. Some few pus cells were found in the urine at this time, but not enough to indicate a pyelonephritis. The lungs remained clear, though mucus and some blood appeared at times in the stools, indicating a condition of the colon which affected the temperature in some degree. Three days after the second operation the autogenous vaccine was ready for use, the first culture having failed to grow with sufficient activity to provide a vaccine.

Some reaction followed the inoculation of the vaccine, the temperature even rising to 105° the second day after its use. From this point on, a rapid subsidence of the temperature took place. On April 24th, a small skin abscess of the scalp which had newly developed, with a slight rise of temperature, was incised. Both wounds are doing well.

Case 3.—A tall girl of fourteen years, large for her age, always rather delicate; has grown very rapidly for the past two years. Her mastoiditis of four days' duration followed a severe attack of tonsillitis, accompanied by profuse discharge from the ear, extreme tenderness of mastoid and high temperature.

At operation (January 10th), an extensive edema existed in the tissues over the mastoid. Blood stained serum was encountered throughout the diploetic mastoid. Free pus was found directly in contact with the lateral sinus, which was exposed externally and anteriorly for a length of three-fourths of an inch, but was apparently healthy. After the operation the temperature rose but once over 101° F., but the patient complained greatly of pain in her limbs and back. This continued to be the cause of much complaint, and on January 14th one-half cc. of an autogenous streptococcic vaccine, containing 50 million bacteria to the cc., prepared by Dr. Murray, was given. The afternoon following the first dose the temperature rose to 101.2,° with an aggravation of the pain in the limbs and back. The next day she was much more comfortable than before the inoculation, the pulse rate became slower and stronger by the second day, and on the third day the pain had left the limbs and she seemed brighter and more comfortable. Throughout her convalescence, which was rather prolonged, more or less tenderness of the joints persisted, and a mild attack of chorea occurred about the time of the complete healing of the mastoid wound. I am inclined to believe that these symptoms would have been in less degree if we had persisted in the use of the vaccine.

Case 4.—Harry S., age nine months, a delicate looking, wasted infant of mulatto (West Indian) parentage, was received at the Brooklyn Eye and Ear Hospital for operation. There was a tense swelling above and behind the right ear. A purulent discharge from the ear had existed for an indefinite period. Operation was performed November 19, 1912. On incision, much pus was liberated, besides which the swelling contained flaky fibrinous masses of a cheesy consistency, which were curetted away. A sinus in the bone led to the antrum. The malleus and incus, which were removed, appeared necrotic. A small area of healthy looking dura was exposed above and posteriorly. Two weeks later a swelling appeared on the opposite (left) mastoid region, and on December 5th this was also operated upon by Dr. Bouvier, senior house surgeon of the hospital, who reported that thick curd-like flakes of pus similar to the condition found in the right had been likewise found in this (the left) side. Neither wound made the least progress toward healthy granulating or healing. Mercurial inunctions were prescribed, but an irregular septic temperature developed, and the child became still more emaciated. A polyvalent vaccine (Van Cott's) was administered every fourth day for a period of four weeks. The child died February 6, 1913, having been in the hospital ten weeks. Dr. Murray performed an autopsy. Both mastoid wounds were clean, but almost as little healed as when operated upon. The brain was entirely normal and uninfected. The dura was exposed over three small areas, two on the right, one on the left side. Both lungs were extensively consolidated. A pleurisy of a serofibrinous type was present, the fibrin being deposited in thick layers. A very large abscess was found in the middle of the right lung. This was the only fatal case in the series.

Case 5.—Radical mastoid for chronic suppuration, infection of the labyrinth. A boy of eleven years was operated on November 15th, for chronic suppuration of the left ear, existent since infancy. A radical mastoid operation was performed. The incus was absent. Vomiting persisted for three days after operation. No nystagmus, tongue coated. Calomel was ordered, after which the bowels moved and the nausea ceased. Two days later (21st) the temperature rose to 102° F. (mouth), and persistent vomiting recurred, especially on rising in bed. Vertigo as if falling forward (not toward the affected side) and severe headache. Slept but little. Next day more comfortable. Mind perfectly clear. Some stiffness of neck, temperature remitted, but again rose to 102° in the afternoon. Polyvalent vaccine one-half cc. was administered every four days, the temperature continuing irregular for a period of three weeks, meanwhile assuming a flatter curve and a gradual cessation of all untoward symptoms. Ear entirely healed and dry at the end of nine weeks, but hearing nil in this ear.

Case 6.—A man, thirty-six years of age, with marked mastoid tenderness and severe pain in left ear; for six days profuse discharge from left ear, with a history of previous ear trouble. Operated February 27, 1913, the culture from mastoid antrum was reported by Dr. Murray to be pneumococcus. The next day he complained of severe pain in the right side of the chest, especially on deep breathing. Dr. Ritter, senior house surgeon, applied wide bands of adhesive plaster. The temperature on the next day (March 1st) rose to 104.4.° No consolidation could be detected. On the next day, Dr. Frank L. Tucker examined the chest at my request, and found a pneumonia developing, an area of consolidation in the right lung being clearly made out. On March 3rd, and for three days thereafter, a stock pneumovaccine was administered daily, each dose representing 40,000,000 bacteria. On March 4th the temperature came down to 99.4,° with profuse perspiration, and did not after rise above 101.° Recovery rapid and without further complications.

LXVIII.

ON THE EFFORTS OF NATURE TO CURE SEPTIC THROMBOSIS OF THE LATERAL SINUS.*

By Harry Friedenwald, M. D.,

Baltimore.

Two cases have come under my observation which impressed me with the curative efforts of nature—if we may so call the processes which tend to circumscribe and eliminate the infected thrombus in the lateral sinus. When we consider the desperate prognosis of untreated cases, we are warranted in giving our attention to these observations, together with those that have been reported hitherto. In doing so there is not the slightest intention to minimize the danger of untreated thrombosis.

Case 1 has already been reported in another connection to this society.[1] A brief statement is as follows:

In December, 1913, I was called to see a child, two years of age, who had within a few months suffered from an attack of scarlet fever, followed by measles. Double purulent otitis media was the result. A swelling had appeared over the left mastoid, which a physician had incised three weeks before I saw the patient. I found a marked hard swelling, about an inch behind the incision, over the mastoid. The ears were both discharging. On the following day the mastoid operation was performed. No visible opening into the mastoid was found in the surface of the bone, which contained pus and granulations. The mastoid process having been cleaned out, a second incision was made, one inch perteriorly, into the abscess, and much pus evacuated. The bone was denuded. The patient made a good recovery, but both wounds continued to suppurate, and excessive granulations formed. In dressing the wounds we found that a probe could be passed far into the posterior one, but it was not possible to determine its exact position.

---

*Read before the American Laryngological, Rhinological and Otological Society, May 9, 1913.

On February 3rd, the patient having been admitted into the City Hospital (Baltimore), I found that the probe passed far into the skull cavity. An incision was made which joined the two previous incisions and the bone was exposed. It was then found that a fistula extended from the posterior wound into the skull, the opening being situated in the mastooccipital suture. The opening was not more than five or six mm. in diameter and perfectly circular. It was enlarged and the bone removed forwards towards the ear, thus exposing the fistulous canal. We then found that the pus had not come from an extradural abscess, but that it was the lateral sinus itself which was continuous with the fistula. A grooved director was passed into the canal and it was slit open. In the sigmoid portion the part was found where the sinus had been exposed to the mastoid disease and where the infection had occurred. It was evident that a septic thrombus had formed, but had been securely shut off from the circulation by firm clots, and that the pus had evacuated itself through the emissary vein, thus producing the abscess.

The case just described furnished an example of the limitation of the septic thrombus which is to be regarded as a conservative effort of nature.

Case 2.—Katie S., aged eight years, was admitted to the Mercy Hospital (Baltimore), November 20, 1912. She had had measles when two years old, but not scarlet fever. The right ear had been discharging since she was five years old, the discharge having a bad odor for some time. When the discharge was free there was no pain, but when the ear was not running she complained of great pain in the head. For about a year there were frequent recurrences. The pain would last a few hours and would then pass off. She was able to go to school until four days before entering the hospital. At this time she became quick sick, and a physician was called in, who thought she had typhoid. She had high fever. was delirious and very weak.

On entering the hospital we found destruction of the right tympanic membrane and the middle ear filled with foul pus. There was no marked sensitiveness over the mastoid or over the course of the jugular vein. Her temperature showed a pyemic character, with mild chills, high rises and rapid falls of temperature, as follows:

November 20th, 4 p. m., 100.4°; 8 p. m., 100.8°; 12 m., 105.4°.

November 21st, 4 a. m., 103°; 8 a. m., 102.4°; 12 n., 98.8°; 4 p. m., 98°; 8 p. m., 99°; 12 m., —.

November 22nd, 4 a. m., 99.6°; 8 a. m., 97.6°; 12 n., 97.8°; 4 p. m., 97.8°; 8 p. m., 98°; 12 m., 101.8°.

November 23rd, 4 a. m., 99°; 8 a. m., 98°; 12 n., 97°; 4 p. m., 97.8°; 8 p. m., 101.8°; 12 m., —.

November 24th, 4 a. m., 102°; 8 a. m., 100.4°; 12 n., 98.2°.

The urine showed some albumin and a few hyalin and granular casts and pus cells. The blood examination revealed polynuclear 76 per cent, small mononuclear 17 per cent, large mononuclear 6 per cent, and a total blood count of 20,500. A blood culture made on November 23rd, on agar plate, bouillon and serum water, was sterile. The eyegrounds were normal.

The operation was performed November 24, 1912. Ether anesthesia. The regular mastoid incision was made, extending from the level of the top of auricle down to a point below the tip of the mastoid. The bone was exposed widely on both sides. A minute fistulous opening was found above the mastoid antrum, from which pus of a very foul odor exuded. This opening was enlarged with chisels; the bone was very sclerotic; a large cavity was entered, filled with the same character of pus, pulsating actively. The sinus was found exposed extensively. The antrum was then opened and contained a cholesteatoma. A radical mastoid operation was performed, thoroughly cleaning the middle ear of pus and cholesteatomatous masses. The bone over the sinus was next removed upward to the knee and downward to the bulb. The upper half of the sigmoid sinus appeared distended and covered with coarse, thick granulations. The lower half was smooth, whitish and seemed collapsed. On close examination the entire wall of this part of the sinus was seen to have been destroyed over a distance of about 2 cm. Venous blood flowed freely from the upper and after-incision. A probe was introduced and, when followed down a short distance, showed firm closure of the sinus and demonstrated the complete destruction of outer wall below. Bleeding was stopped with pressure. At the lower end an opening was found entering the sinus for a short distance and discharging pus. For evident reasons the jugular was not tied. A plastic was made by splitting the posterior part of the canal lengthwise, with incisions downward and upward in the form of a "Y." The canal was packed and a drain placed into the lower end of the sinus, and the wound was dressed as usual. From the pus taken from the mastoid wound, short chain streptococci developed in pure culture.

The temperature after the operation became normal and at no time rose to 100.° The patient made an uneventful recovery and was discharged from the hospital January 2, 1913, the wound almost completely healed.

In this case it is seen that the sinus was completely and firmly closed at both ends, and that the thrombosed portion had relieved itself entirely by destruction of almost the entire outer wall of the affected portion.

These cases brought to my mind others in which complete closure was found at one end of the thrombosed sinus—or even at both ends; in the latter case so firm as to convert the diseased part of the sinus into a tensely filled pus sac.

A number of cases have been reported in which complete obliteration of the sinus by collapse or, more probably, by organized thrombus has been observed, either during the operation or after death. Thus Griesinger[2] described a postmortem case of complete obliteration of the entire sigmoid sinus by a firm thrombus. Schwartze[3] published a like case in 1873. Similar cases were also published by Alt,[4] Politzer,[5] and Zaufal.[6] Torök[7] reported a case in which there was injury of the sinus during an operation. Six months later, pyemic symptoms; the sinus was exposed and found to contain, from the tip of the mastoid to the knee, an organized thrombus of firm connective tissue. At the autopsy the obliteration was found to extend to the torcular. Purulent phlebitis had developed beyond this and had been the cause of death. Grüning[8] found destruction of the wall of the sigmoid sinus and a perisinus abscess and the peripheral portion of the sigmoid sinus converted into a solid cord. Barr,[9] in a case of temporosphenoidal abscess, found "complete occlusion of the sigmoid sinus, the walls of which were converted into a fibrous cord." Suckstorff and Henrici[10] report a case of spontaneous cure of thrombosis; at the autopsy the transveres sinus was found to be completely obliterated by connective tissue. The thrombosis had run a latent course.

A number of cases are reported of thrombosis running a nonfebrile course in which obliteration of the sinus took place.[11]

A number of these cases presented fistulous openings in the sinus walls. The nonfebrile course is variously explained—by the aseptic character of the thrombus, by the mild nature of the infection or by the discharge of pus when formed through an opening in the external wall.

We cannot determine with certainty how the obliteration takes place—whether by direct organization of the thrombus without its disintegrating, by emptying of the pus formed through the outer wall, or by collapse and agglutination of the walls.

A large number of cases bearing marked similarity to those reported by me are to be found in our literature. Hoffmann[12] reports one case in which the central and peripheral ends of the thrombosed sigmoid sinus at the operation were found to be closed by connective tissue obliteration of the sigmoid sinus[13] in a child with extensive defect of the inner wall of the mastoid process and extradural abscess. On splitting the sinus, it was found firmly closed by organized thrombus with but a little of the lumen retained. The fever subsided immediately. Wernicke[14] found the sigmoid converted into a 2.5 cm. long and 1.0 cm. wide abscess, completely shut off above and below by organized masses. In a second case the obliteration existed only toward the central end. These cases were cured by operation. Wernicke gives a complete bibliography of the early cases up to 1900. Streit[15] reports a case of spontaneous cure of thrombosis by obliteration of sinus. There were no symptoms of sinus thrombosis. The operation was done for mastoiditis and exposed a cavity whose inner wall was covered with granulations and was found to be the obliterated sinus. Hölscher[16] reports one case in which all symptoms of thrombosis were absent, while in the second there were rigors and swelling in the neighborhood of the emissary veins, but otherwise only headaches and slight evening temperature. On opening, a perisinus abscess was found; the sigmoid sinus was absent in both cases, being replaced by granulation tissue which passed over directly posteriorly into the normal sinus. He reports a third case of death from cerebellar abscess without symptoms of sinus thrombosis; no sinus was found at the operation. At the postmortem the transverse sinus was found enlarged from torcular to knee, and from this point on completely closed by a firm thrombus. Bárány[17] describes a case of spontaneous cure of sinus and bulb thrombosis. The case was one of mastoiditis with extensive subperiosteal abscess. On opening the mastoid the sinus and dura were found extensively exposed, greatly thickened, and in the neighborhood of the bulb a black opening was found in the sinus through which a sound could be entered for a short distance in both directions. The sinus was thin and obliterated. Recovery took place without further operation. In the discussion Urbantschitsch[18] stated that he had demonstrated a similar case of cured sinus thrombosis with obliteration of the lumen. On exposing the sinus in a case of putrid cholesteatoma, a tablespoon of foul green pus was discharged, the wall of the sinus, which was discolored, was slit up; the whole sinus was found filled with nonpurulent thrombotic masses. Toward the sinus transversus the sinus walls were completely adherent to each other.

Recent experimental studies of this subject have brought interesting results. Haymann[19] was able to produce and study sinus thrombosis and pyemia in dogs and monkeys. Among his conclusions the following are to be specially mentioned: The thrombi in their earliest stages may contain microorganisms, or the latter may enter the thrombi subsequently from without or be brought to the thrombi by the blood stream. The number of organisms contained in the thrombus vary greatly, but the ends of the thrombus are usually most benign. A moderate number of organisms does not prevent organization of the thrombus. Experimental infected thrombosis shows a marked tendency to spontaneous recovery; and even malignant forms, in which the only wall was destroyed, at times showed organization, vascularization and recanalization of the sinus.

In a subsequent paper Haymann[20] considers the processes tending to spontaneous cure of sinus thrombosis in man. He reports a case of purulent otitis media for a year with death from meningitis. The sigmoid sinus was obliterated throughout a large part, completely closed by firmly organized connective tissue with distinct evidences of beginning recanalization, a condition similar to his findings in experimental thrombosis. He insists that the thrombosis must not be looked upon as noninfected because of the spontaneous recovery, though the infection may not have been very virulent. This conclusion is supported by his experimental observations. This is the first case in which histologic proof is brought of complete spontaneous cure of a sinus thrombosis of otitic origin. He refers to the case of Miodowski,[21] in which there were similar histologic findings, but points out that though the jugular and sinuses were thrombosed, the infection was from a cervical gland and therefore not otitic. He concludes that processes tending to spontaneous recovery in sinus thrombosis in man are not of rare occurrence. The recovery may be so complete that recanalization is established.

Voss and Haymann have emphasized the importance of these processes in their practical bearings. Voss,[22] in a paper bearing the startling title "Lasst den Thrombus in Ruh," warns against the removal of the thrombus with the sharp spoon; he points out that the entire outer wall of the diseased part should be carefully removed, and he only incises the thrombus. The practice to leave the firm ends undisturbed would appear to be in accord with the experience which the cases described and referred to afford. A source of danger which must be emphasized is that of incising the collapsed sinus or the inner sinus wall when the outer wall has been destroyed, with the intent of opening the sinus. Thus Grünert and Dallmann[23] reported a case in which the sinus became obliterated after injury during operation, and an incision through the fibrous cord passed directly into the cerebellum. In another case, Uchermann[24] in this manner opened a cerebellar abscess by incising through a collapsed lateral sinus. And Herzfeld[25] had a similar experience in a case of cholesteatoma. He opened into a sinus, the outer wall of which was covered with granulations, and incised directly through the obliterated sinus into the brain. The patient recovered.

The firm closure of both ends of the thrombus with breaking down in the middle is less apt to lead to metatasis and general infection. But, as Hessler[26] has shown, the advantage is not entirely on this side, for those cases in which general infection occurs are less apt to develop cerebral abscess and meningitis, and vice versa.

Another, though remote, source of danger was pointed out by Hoffmann,[27] namely, that an organized thrombus may subsequently disintegrate and lead to grave results. Brieger[28] reported a case of such reinfection of a healed sinus and jugular thrombosis.

We are warranted in concluding that the course of a thrombosis varies according to the virulence of the infection and other influences. It may become organized without disintegration, and the lumen may even become reestablished. But even in those cases in which it becomes purulent in its central part, the firm closure of both ends and the formation of a fistula or the entire destruction of the outer wall may permit its more or less successful drainage, and thus tend to its spontaneous cure. This process in its nature is the same that takes place in every case in which conditions are brought about by operation which permit the formation of a noninfected thrombus at both ends of the infected portion of the sinus. It is apt to be incomplete when spontaneous, since the anatomic structure of the temporal bone places many obstacles in the way of its complete and easy drainage.

REFERENCES.

1. The Laryngoscope, January, 1909, p. 22.
2. Griesinger: Arch. der Heilkunde, 1862.
3. Schwartze: Arch. f. Ohr., Vol. 13, p. 98.
4. Alt: Verhandl. d. oesterreich otol. Gesellsch., October, 1905.
5. Politzer: Textbook.
6. Zaufal: Arch. f. Ohr., Vol. 17.
7. Torök: Arch. f. Ohr., Vol. 50, p. 65.
8. Grüning: Trans. Amer. Otol. Soc., Vol. 35, p. 78.
9. Barr: Textbook, fourth edition, p. 330.
10. Suckstorff and Henrici: Zeitsch. f. Ohr., Vol. 44, p. 159.
11. Alexander: Arch. f. Ohr., Vol. 64, p. 89.
    Uchermann: Arch. f. Ohr., Vol. 72, p. 263.
    Blau: Arch. f. Ohr., Vol. 74, p. 200.
    Nager: Zeitsch. f. Ohr., Vol. 53, p. 162.
    Voss: Zeitsch. f. Ohr., Vol. 53, p. 316.
    Uffenorde: Monatssch. f. Ohr., Vol. 41, p. 661.
    Wild: Zeitsch. f. Ohr., Vol. 57, p. 247.
    Seidler: Arch. f. Ohr., Vol. 81, p. 177.
    Bondi: Monatssch. f. Ohr., Vol. 43, p. 641.
    Buys: Arch. Internat. de Laryng., Vol. 27, p. 127.
    Welty: Calif. State Journal Med., February, 1908.
    Auerbach: Laryngoscope, December, 1912.
12. Hoffmann: Arch. f. Ohr., Vol. 45, p. 131.
13. Hoffmann: Arch. f. Ohr., Vol. 50, p. 76.
14. Wernicke: Arch. f. Ohr., Vol. 48, p. 197.
15. Streit: Arch. f. Ohr., Vol. 56, p. 212.

16. Hölscher: Bresgen's Zwang. Abhandl., Vol. 6, 1902.
17. Bárány: Monatsschr. f. Ohr., Vol. 43, p. 790.
18. Urbantschitsch: Monatsschr. f. Ohr., Vol. 43, p. 182.
19. Haymann: Arch. f. Ohr., Vol. 83, p. 1, 1910.
20. Haymann: Muench. med. Woch., Vol. 58, p. 2443, 1911.
21. Miodowski: Arch. f. Ohr., Vol. 74, p. 335.
22. Voss: Zeitsch. f. Ohr., Vol. 53, p. 316.
23. Dallmann: Arch. f. Ohr., Vol. 65, p. 127.
24. Uchermann: Zeitsch. f. Ohr., Vol. 46, p. 313.
25. Herzfeld: Zeitsch. f. Ohr., Vol. 2, p. 141, 1909.
26. Hessler: Otitische Pyämie, p. 274.
27. Hoffmann: Verhandl. d. deutsch. otol. Gesellsch., 1897, p. 132.
28. Brieger: Zeitsch. f. Ohr., Vol 5, p. 29.

LXIX.

BONE CONDUCTION IN SYPHILIS.*

By Dr. Oscar Beck,

Vienna.

In Stuttgart, during a discussion of a paper by Voss, I made a preliminary report of my investigations concerning bone conduction in syphilis, which subject I should like to take up more in detail in this article.

During the examination of a great number of patients infected with syphilis, who at the same time suffered from disease of the ear, I observed that bone conduction was strikingly reduced in comparison to the shortening of air conduction in affections of the internal ear. It was very evident, also, that lengthening of bone conduction frequently could not be so distinctly elicited in lesions of the sound conducting apparatus as we are accustomed to obtain it in nonluetic patients troubled with a similar ear condition. Since, however, all our methods of measuring bone conduction are liable to subjective variation, and since there was the possibility of a lengthened bone conduction in the examiner (in this instance myself), I had my results controlled by other examiners, whose findings did not differ materially from mine. This shortened bone conduction in syphilitic affection of the ear is mentioned in several places, as I found in a subsequent search of the literature. But the almost regular occurrence of this symptom and its practical value in diagnosis, especially in the differential diagnosis between luetic and nonluetic diseases of the inner ear, so far have not received their proper recognition, although Wanner, during the discussion in Stuttgart, stated that for a long time, on the basis of this symptom, he had been able to diagnose syphilis.

In the first place, I deemed it necessary to make a careful study of bone conduction in syphilitics who complained of no ear symptoms, and in whom an otoscopic examination revealed normal conditions. The method which I employed for this purpose was originally quite crude. I applied a vibrating tuning fork of middle register to the mastoid process of the patient, and when it ceased to be heard by him, I immediately transferred it to a similar position on the mastoid bone of a nonluetic person with normal hearing.

---

*Translated by E. Roth, M. D.

In spite of the fact that this method has many chances for error, I was able in the overwhelming majority of cases to demonstrate that differences exist between the bone conduction of normal hearing luetics and the bone conduction of normal hearing nonluetics. I examined patients with all kinds of physical manifestations of syphilis, particularly those of the second stage. Examinations were made also of patients with latent syphilis, whose history and positive Wassermann reaction proved the existence of the disease. In this series of cases were included also patients who had been infected with syphilis, but who, after various courses of treatment, gave a negative serum reaction.

The table, which was prepared at my suggestion by Dr. A. W. Hilger of St. Paul, Minnesota, may now be referred to. In this collection no cases were selected, the findings being the results obtained in fifty-six patients sent to our clinic for otologic examination according to the usual routine before the administration of salvarsan.

From this table, which was prepared two and one-half years ago, it would appear that only in a small percentage of cases is the shortened bone conduction not found—which is a fact. Since then, however, I have observed larger series of cases, and in these (as is to be expected when a large number of cases is studied) the percentages varied materially. Patients with eruptive manifestations of syphilis are found in whom normal bone conduction is elicited, and the same is true of patients with tertiary symptoms. In cerebral syphilis, which provokes symptoms of various cranial nerve involvement, the shortening is practically never absent. In the metaluetic types of the disease, such as tabes or paresis, the symptom must be cautiously used. For here we are dealing mostly with subjects on the further border of middle age, in whom, independent of the disposition to arteriosclerosis produced by syphilis, phys-

No.	Name.	Age.	Duration of Lues	Eruption	Otoscopic Findings	Aural Symptoms	Air Conduction	Bone Conduction*
1	E. Z.	45	6 months	—	Normal	Never	Normal	R. 5; L. 5
2	A. J.	32	3 weeks	—	Normal		Normal	R. 8; L. 10
3	A. D.	28	3 weeks	—	Normal		Normal	R. 8; L. 13
4	K. N.	49	3 weeks	—	Normal		Normal	R. 7; L. 11
5	C. E.	30	6 weeks	+	Normal		Normal	R. 12; L. 5
6	J. H.	33	5 months	+	Atrophy L. and R.		Normal	Normal
7	D. D.	—	3 years	+	Normal		Normal	R. 17; L. 30
8	K. M.	20	6 weeks	—	Normal		Normal	Normal
9	J. N.	26	5 weeks	+	Normal	Noises	Normal	R. 8; L. 10
10	B. G.	26	7 years	+	Chr. supp. L. and R.		Shortened	Normal
11	J. P.	34	6 weeks	+	Normal		Normal	R. 14; L. 10
12	F. J.	37	5 years	+	L. scar; R. chr. adhesive processes	Noises	Normal	R. 7; L. 10
13	M. Z.	29	8 weeks	—	R. and L. cicatrices	R. and L., Discharge during childhood	Normal	Normal
14	A. N.	37	2 weeks	—	R. norm.; L. O. M. S. C.	L., Discharge	R., Norm.; L., Short	R. 4; L. norm.

* The figures show the amount of shortening in seconds.

No.	Name.	Age.	Duration of Lues	Erup-tion	Otoscopic Findings	Aural Symptoms	Air Conduction	Bone Conduction*
15	J. W.	21	3 weeks	—	Normal		Normal	R. 12; L. 15
16	F. W.	18	5 days	+	Normal		Normal	Normal
17	M. L.	20	1 year	—	Radical Op. Right		R., Short'd; L. Norm.	R. 5; L. 8
18	T. S.	19	8 days	+	R. and L., O. M. S. C.	R. and L., Discharge	R. 10; L. 20	R. & L. length'd
19	F. H.	28	8 years	—	Normal	Normal	Normal	R. 15; L. 8
20	G. H.	28	2 months	—	Normal	Normal	Normal	R. 15; L. 12
21	L. K.	36	3 weeks	+	R., Drum destroyed; L., Cicatrices	R. and L., Discharge	R., Norm.; L. 50	R. 17; L. norm.
22	M. P.	25	4 weeks	+			Normal	R. 14; L. 8
23	J. H.	26	2 months	+			Normal	R. 17; L. 14
24	F. E.	20	8 days	—			Normal	R. 11; L. 8
25	R. R.	21	2 months	+			Normal	R. 13; L. 11
26	K. B.	36	12 years	+			Normal	R. 18; L. 15
27	H. P.	31	3 weeks	—			Normal	R. 5; L. 3
28	J. S.	42	3 weeks	+			Normal	Normal

29	B. R.	23	1 year	—?			Normal	R. 17; L. 9
30	H. S.	23	6 weeks	+			Normal	R. 13; L. 7
31	K. S.	24	3 weeks	—	R., Cicatrix; L., Adhesive processes	Previously	Normal	R. norm.; L. 13
32	K. R.	26	3 weeks	—			Normal	R. 5; L 10
33	F. T.	30	4 weeks	—			Normal	Normal
34	V. B.	25	4 months	+	Scars R. and L.	Impaired hearing R. and L.	R. and L., Shortened	R. 10; L. 12
35	R. S.	23	2 years	+	L., Chr. supp. healed.		R., Norm.; L., Short'd	R. 10; L. 5
36	J. M.	28	5 years	+			Normal	R. norm.; L. 5
37	A. W.	19	34 days	—			Normal	R. 10; L. 10
38	E. G.	23	5½ mos.	—	Normal		Normal	R. 17; L. 28
39	J. S.	31	3 weeks	—	Normal		Normal	Normal
40	A. H.	75	1 year	+	Normal		Normal	R. 5; L. 10
41	A. H.	28	3 years	+	Normal		Normal	Normal
42	J. M.	51	14 years	+	Normal	Vertigo and noises	Normal	R. 10; L. 15
43	A. A.	20	6 years	+	Normal		Normal	R. 10; L. 16

* The figures show the amount of shortening in seconds.

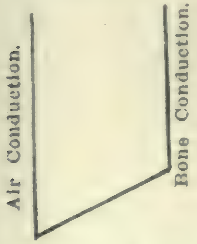
No.	Name.	Age.	Duration of Lues	Erup-tion	Otoscopic Findings	Aural Symptoms	Air Conduction	Bone Conduction*
44	J. R.	30	1 year	+	Retracted drums		Normal	Normal
45	L. L.	26	3 weeks	+	Normal		Normal	Normal
46	A. S.	32	3 weeks	—	Normal		Normal	Normal
47	J. S.	36	1 year	+	Normal		Normal	R. 5; L. 10
48	A. M.	27	1 month	—	Normal		Normal	R. 5; L. 5
49	H. H.	25	3 weeks	—	Normal		Normal	Normal
50	L. A.	20	2 weeks	—	Normal		Normal	Normal
51	M. Z.	32	6 weeks	+	Normal		Normal	R. 2; L. 6
52	R. F.	23	3 years	+	Normal		Normal	R. 15; L. 15
53	F. V.	19	8 weeks	+	Normal		Normal	R. 10; L. 10
54	J. S.	19	2 months	+	Normal		Normal	R. 15; L. 10
55	R. E.	15	6 weeks	—	Normal		Normal	R. 6; L. 6
56	M. J.	30	2 months	+	R. and L., Dry perf.	Impaired hearing	R. and L., Shortened	R. and L. norm.

* The figures show the amount of shortening in seconds.

iologic changes in the vessels are already present, leading to a shortened bone conduction.

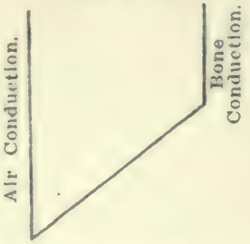
The following fact is evident: Although there are no exact and systematic tests which express mathematically the relationship between air and bone conduction in the normal and in the impaired ear, still we were convinced during our examination that bone conduction was decidedly reduced in luetic affection of the ear, and that a similar degree of shortening corresponded to a greater impairment of hearing in an ear whose internal ear was not affected by syphilis.

Figure 1-a.



Læsio auris internæ non-luetica.

Figure 1-b.



Læsio auris internæ luetica.

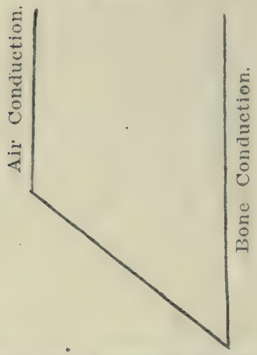
Naturally there are differences depending on the interpretation of each examiner, as mentioned above.

Of interest and of diagnostic value are the results of the tuning fork in patients with middle ear disease who have had syphilis. I shall briefly relate a case of this kind.

A young woman came to our clinic, complaining of various febrile symptoms, intense headache, and severe pulsating pain in the ear, which had lasted several days.

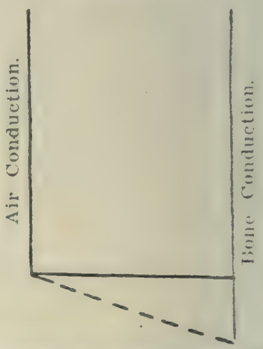
The drum membrane was diffusely reddened and swollen. No details were visible and the posterosuperior quadrant of the drum was bulging. Weber was lateralized to the affected ear, in which there was decided impairment of hearing. Air conduction was almost normal, while bone conduction in the bad ear, tested with a tuning fork of middle register, on the mastoid process, was surely not lengthened. I made a diagnosis of "acute middle ear suppuration, very probably in a syphilitic individual." The patient admitted the infection and the Wassermann was very positive. I was the more convinced of the presence of syphilis because the bone conduction was not lengthened, in spite of the fact that an acute otitis media was present. Therefore, there must have been a shortened bone conduction in the ear prior to the advent of the acute otitis; and the increased bone conduction consequential to the acute middle ear affection altered the picture from a shortened bone conduction to one of normal duration. To this assumption exception is naturally to be made in those cases where a shortened bone conduction is attributable to an affection of the internal ear, resulting from some injurious agent or due to a harmful vocation.

Figure 2-a.



Impediment to sound conduction in a non-luetic individual.

Figure 2-b.



Impediment to sound conduction in a luetic individual.

The question still remains to be answered as to the point of time after syphilitic infection at which this symptom, if it occurs at all, is elicited.

In very early cases presenting an initial lesion without local glandular involvement, and having a negative Wassermann, i. e., in patients in whom the diagnosis was based solely on the demonstration of spirochetæ in the primary sore, I have never observed a shortening. From these I have excluded those cases showing shortened bone conduction whose histories gave other etiologic factors. But in these cases shortened bone conduction is generally connected with an impairment of hearing. The symptom makes its appearance about the time when the local syphilis becomes general, approximately with the occurrence of the first papular rash; occasionally during the period when Wassermann becomes positive. The exact time cannot be fixed, for among my patients there are those who, with normal hearing and without other demonstrable cause, showed a shortened bone conduction even before the serum reaction became positive; so that this symptom could be the first to awken the suspicion of a generalized syphilis. Patients were examined, also, who at the beginning of the eruption still had normal bone conduction and in whom a shortening was observed only after the rash had existed for quite some time.

On the other hand, a mechanic in whom a chancre was diagnosed after the demonstration of spirochetæ in the initial lesion, was sent to me for otologic examination preparatory to the injection of salvarsan. Wassermann negative; no glandular swelling or other sign of general syphilis. Since the bone conduction on both sides was shortened, although the patient had normal hearing, I believed, in opposition to the dermatologist, that I could made a diagnosis of already generalized syphilis.

The patient was treated with salvarsan and Hg., and for half a year there were neither clinical symptoms nor blood findings which pointed to syphilis. Bone conduction remained shortened. After six months papules appeared on the lips and tonsils, and Wassermann became positive. This case seems to indicate that shortened bone conduction in the presence of normal hearing may precede every other symptom of generalized lues.

I could never note any difference in the shortening before and after salvarsan or mercurial treatment. It is by no means easy to give a true explanation for this interesting condition. At the outset I thought I could find a connection between this symptom and a change in the blood pressure, but all experiments in this direction gave negative results. Consequently, it seemed necessary to determine whether shortened bone conduction was not to be found under other circumstances in individuals with unimpaired hearing. As is known, Wanner has claimed shortening of bone conduction to be characteristic of pathologic changes in the cranium or its contents. We know, also, that in tumors and in hydrocephalus, even without cochlear or vestibular symptoms, shortened bone conduction may be found. Such an observation was referred to by Ruttin in the Austrian Otological Society.

Herzog, in a recently published article in the *Münchener* *med. Wochenschrift,* showed that in cases of growths of the dura to the bone, bone conduction could remain unaltered; indeed, it could be lengthened under certain circumstances. Besides in cases of tumor, hydrocephalus, and cerebral arteriosclerosis (Konrad Stein), I myself have been able to find this symptom unquestionably present in three cases of epilepsy and in one case of tetany. These observations, therefore, led to the conclusion that shortened bone conduction in the presence of normal hearing is indicative of a symptom which cannot be directly attributed to the ear itself, but is the result of the increased intracranial or intraspinal pressure.

In almost all of the recently published works dealing with the examination of the cerebrospinal fluid and the determination of its pressure in syphilis, a rise in the pressure was found by the respective authors. I have, therefore, in several cases measured the shortening of bone conduction before and after lumbar puncture. With the exception of one case, it was possible within three or four hours after lumbar puncture to establish the disappearance of the shortened bone conduction. I withdraw on the average five to six cubic centimeters. It was remarkable that in one case the shortening was greater two hours after the lumbar puncture than it had been prior thereto, and that only after another two hours did this shortening disappear. However, two or three days later the shortening was again to be found.

In spite of the fact that in this direction my investigations have not been very extensive, I believe that the shortening of bone conduction is to be attributed to the increase of the intracranial pressure, and that those cases of lues which do not present the symptom also show no increased cerebrospinal pressure.

If we admit this causative relationship between shortening of bone conduction and a rise in intracranial pressure, it also becomes clear why this symptom is as a rule first found when local syphilis becomes general. In the same manner may be explained the vertigo and disturbances of equilibrium during the second stage of florid lues, concerning which I made a communication at the Otological Congress at Frankfurt on Main. On this ground also may be understood the exaggeration of skin and tendon reflexes which Finger established years ago on the basis of fifty cases examined immediately before and after the eruption of the rash. Remarkable, and especially worthy of mention, is the circumstance that occasionally we may determine a difference in the degree of shortening between the two sides of the same patient; that, for example, the shortened bone conduction of the right mastoid may be decidedly greater than that of the left mastoid.

I should like to describe briefly a particularly characteristic case:

T. M., twenty-five years old, kitchen maid. Three weeks ago initial lesion on the genitalia. Pale roseola on the trunk and extremities. Numerous large papules on the labia and at the anus. Lymphadenitis inguinalis. Wassermann positive.

Otoscopy.—Drum membrane and middle ear on both sides, normal. Bone conduction, measured with the electric tuning fork of Urbantschitsch, twice as short on the right side as on the left.

Nervous system.—On the left side distinct exaggeration of reflexes.

In this case, therefore, the greater shortening—that is, the greater intracranial pressure of the right side—is parallel with the exaggeration of the contralateral reflexes, a status which again points to the connection between intracranial pressure and shortening.

My chief, Prof. Urbantschitsch, has devised a tuning fork, which by the application of the electric current enables us to measure bone conduction exactly and to express it numerically. Prof. Urbantschitsch was kind enough to measure the bone conduction in a great number of luetics, and we were able here also in 80 per cent of the cases to confirm the above described findings. The exact construction of this instrument will be demonstrated by my chief on scientists' day in Vienna. In connection therewith I shall report the more exact findings of the difference in bone conduction between luetics and nonluetics, resulting from this method of examination.

LXX.

THE NASAL VOICE WITH REFERENCE TO ITS BEARING ON THE PRACTICE OF RHINO-LARYNGOLOGY.*

By Elmer L. Kenyon, A. B., M. D.,

Chicago.

The special significance of the nasal voice to the rhino-laryngologist lies in this, that its correction is commonly dependent upon nasal or pharyngeal surgery, that under certain circumstances its presence modifies in an important manner the ordinary indications for operative treatment of the nose or nasopharynx, and that, without a knowledge of the nature and significance of the nasal voice, mistakes in diagnosis, prognosis and treatment are liable to be made, and operations of permanent injury to the patient undertaken.

The nature and cause of the nasal voice, or nasaling of the voice, is now, after many years of patient experimental study by men like Passavant, Merkel, Wintrich, Grützner, Schmidt and H. Gutzmann, quite satisfactorily established. One should rule out of mind nasal resonance as distinguished from nasaling, for a certain degree of nasal resonance is a quality of all normal voices. and is particularly significant in all voices of especial richness of quality. Nasaling, on the other hand, is not only due to an extreme exaggeration of the normal mechanical conditions upon which nasal resonance depends, but it is also produced, in the second of its two forms. by an entirely different mechanism from that responsible for nasal resonance. and consequently with a different voice effect.

The familiar nasaling which is produced by obstruction in the nose or in the nasopharynx affects primarily only the normally nasal sounds. altering their quality through impairment of the conditions of resonance in the nose and nasopharynx.

---

*Read before the Chicago Laryngological and Otological Society. May 27. 1913.

When in the production of the nasal sounds, m, n and ng, the vibrating air from the larynx is permitted by the low position of the soft palate to pass outward through an unobstructed nasopharyngeal cavity and through unobstructed nasal cavities, the resulting sounds are normally produced and sound normally nasal. When, however, the nasopharynx or nose is obstructed beyond a certain degree, these nasal sounds, while still nasal, take on an altered quality, sometimes spoken of as a "dead" quality. This alteration from the normal resonance of the nasal sounds produced by obstruction varies as to whether the obstruction be nasal or nasopharyngeal.

The kinds of obstruction in the nose and nasopharynx are known to every rhinologist and need not be considered here. One cause of rhinolalia clausa should, however, be mentioned. When we consider that rhinolalia clausa depends upon obstruction to the free passage of air from the pharynx through the nasopharynx and nose in the production of the nasal sounds, it is evident that such affections of the soft palate as bind the velum closely to the posterior wall must, by preventing the passage of air into the nasopharynx, also be responsible for rhinolalia clausa. This occurs, of course, chiefly from the adhesions following syphilitic diseases of the soft palate.

Rhinolalia clausa involves the speech not only with respect to the nasal sounds themselves, but also affects the sounds immediately preceding or following them, for during the normal production of many of these adjacent sounds the soft palate is still at least partially dropped, and the sounds are, therefore, to a certain degree, nasal. And, besides, the sounds normally oral may be irregularly involved, through their functional nasalization, in the manner to be described later (as rhinolalia aperta functionalis), owing to the exaggeration of the nasal qualities of the speech in general.

The second form of nasaling affects the mechanism for producing not the nasal but the oral sounds. Only one anatomic structure is at fault, the palate. If the palate is normally formed, and if the movements of the soft palate are normal, the velum leaps quickly backward and upward with each oral sound to meet the posterior wall of the pharynx, thus shutting off all or nearly all air from the nasopharynx and nose, and compelling it to pass out of the mouth. Any condition which produces either a permanent opening in the soft or hard palate, or interferes with the movements of the soft palate, has the effect of preventing the relatively complete shutting off of air from the nasopharynx and nose when demanded in the formation of the oral sounds; the sound producing air then escapes freely into the nasopharynx and nose, and consequently transforms the oral into nasal sounds. If the opening from the mouth or pharynx through the hard or soft palate be in the posterior two-thirds of the space between mouth and posterior pharyngeal wall, all spoken sounds are nasal; if the opening occur, on the other hand, only in the anterior third of this distance, all sounds are apt to be normal excepting those normally produced at or near the lips, which then become nasal. In rhinolalia aperta then, when, as ordinarily is the case, dependent upon openings in the posterior two-thirds of the palate, all sounds are organically nasal, the usual nasal sounds m, n and ng being produced normally, and the oral sounds abnormally.

Rhinolalia aperta is dependent characteristically upon cleft palate. Likewise a congenitally short palate, being unable to reach the posterior wall on phonation, has identically the same effect on the voice. Destructive disease of the soft palate, often resulting from late syphilitic ulceration, is a quite common cause. And with the soft and hard palate normally formed, if the movements of the velum be interfered with, rhinolalia aperta follows as a necessary consequenec. This occurs in deep infiltrations of the soft palate, chiefly syphilitic, in organic paralysis, notably bulbar paralysis. in paresis following diphtheria, and in paresis following rough handling of the palate in operations for adenoids.

A combination of the two organic causes of nasaling is not uncommon and complicates the situation. Rhinolalia mixta occurs when the nose or nasopharynx is occluded, and at the same time the palatal closure for the oral sounds is prevented in any of the ways mentioned. The voice partakes of the qualities of both forms of nasaling.

Thus far we have spoken only of organic causes of nasaling; but the functional causes for both forms are of particular importance to the rhinologist, and will not be neglected in a later portion of this article.

The diagnosis of rhinolalia clausa is not difficult for the rhinologist when it depends upon obvious extreme nasal or nasopharyngeal obstruction. Difficulties arise, however, because the degree of obstruction capable of producing nasaling is hard always to estimate, and if one is not on his guard he may be operating unnecessarily to produce enlargement of the nasal or nasopharyngeal space, in order to correct a nasal voice in no way dependent upon occlusion, and of course without improvement of the voice quality following. Such operative procedures may indeed result in a decidedly increased degree of permanent voice impairment. Accurate diagnosis alone can prevent errors of this kind. To this end let the rhinologist determine, first of all, the presence or absence of rhinolalia aperta. This, if present, will direct his attention to the condition of the palate, which should ordinarily have remedial consideration, if impaired, before any existent nasal or nasopharyngeal obstruction.

Rhinolalia aperta may be distinguished quite certainly from rhinolalia clausa by the ear of the rhinologist, if it be sufficiently trained. But ordinarily this method of differential diagnosis is not certain enough. More than twenty years ago Herman Gutzmann devised the following simple but usually effective test for rhinolalia aperta. It is based upon the proven fact that, if the nose be tightly closed while the vowel sounds are being enunciated, the intensity of the nasaling is decidedly increased, whereas if the voice impairment be due to rhinolalia clausa, no change in the quality of the vowel sounds occurs from holding the nose. He has the patient repeat the vowel sounds "ah-e" several times, both with and without the closure of the end of the nose by compression with the thumb and finger, and notes the result in the sound. If there is no change in the quality of the sound the nasaling is not due to rhinolalia aperta, but if the nasaling is thereby increased the test proves the presence of rhinolalia aperta. The test is a delicate one. It is rendered more easily interpreted if the vowels be increased in number. A good series is ah-o-oo-e. This test may be supplemented by one which is perhaps still more delicate, but which demands some experience. The ear-tip at one end of a diagnostic ear tube is replaced by a nasal tip, and the tube is made to connect the naris of the patient with the ear of the physician. By this apparatus all sounds made with a lowered soft palate are grealty intensified, while the oral sounds are heard relatively much less loudly. With the normal voice the nasal sounds and their immediate neighbors are alone intensified; in rhinolalia aperta all sounds, oral as well as nasal, are heard with about the same intensity. In this manner one may listen to spoken sounds directly as they emanate from the nose of the patient; to vowels (see the above series), to the speech of general conversation, or words especially provided for the test. For example, one may use a sentence which in the first half is made up of words each containing a nasal sound, and the last half containing only oral sounds, like the following: "In summer time, when men seem stricken under the strain of the sun, you or I or Frederick will probably be south of the Ohio." Through the nasal ear tube the latter half of the sentence is much less intense than the first half, if the voice be normal, and about equally intense in rhinolalia aperta.

If these tests show that rhinolalia aperta is absent, the nasaling must be due to nasal or nasopharyngeal obstruction. If they show that rhinolalia aperta is present, the diagnosis of any also existing rhinolalia clausa will depend upon the quality of the nasaling and upon the objective evidences of occlusion in the nose and nasopharynx. It is sometimes important, as will be seen later, to learn whether a rhinolalia clausa is dependent not upon obstruction in the nose or nasopharynx, but to an abnormal action of the soft palate. If the nose and nasopharynx are not totally occluded, this can be shown by the diagnostic nasal ear tube, for in such a case the nasal sounds will be greatly reduced in normal intensity, and probably no more intense than the oral sounds.

The application of a knowledge of nasaling in the practice of rhinolaryngology may perhaps be most clearly brought out by the study of certain concrete occurrences. As a preliminary practical step in the discussion, I may mention two recent cases of nasaling, one in a young man and the other in a young woman. The young man was operated for adenoids for no other reason than to improve his voice, but it was not improved by the operation. Upon examination the uppermost part of an adenoid mass was seen to be still present posterior to the choanæ. Rhinolalia aperta was absent. The patient being returned to his physician for reoperation, came back now with a normal voice. The nasaling in the case of the young woman is very marked; the adenoid operation was done three years ago. Here, too, rhinolalia aperta is absent. The amount of adenoid tissue now present is large, and, besides, the nose is markedly occluded. The parents are much more troubled about the voice than about the obstructed breathing, but have not yet consented to return to their physician for further operative treatment, which, from the standpoint of the voice, must include the removal of obstruction in the nose as well as for that in the nasopharynx. The only inference from these cases is the need from the standpoint of the voice for a clean adenoid operation.

But nasopharyngeal operations may have with respect to the voice quite unexpected results. For example, the adenoids in a patient with a markedly nasal voice are completely and expertly removed, and yet the nasaling, both as to quality and intensity, remains unaltered. The nose is found to have but moderate obstruction, this being due to vasomotor swelling. In such a case the rhinologist, if not on his guard (the nasaling being the abnormality demanding correction), may, without further diagnostic study, proceed to operate on the nose. If he does so, this second operation will also be without result in correction of the nasal voice. More than twenty years ago Herman Gutzmann* related a case of exactly this kind occurring in a young German merchant who had gone the rounds of the European capitals seeking relief for an unpleasant nasal voice. Each rhinologist, apparently quite ignorant of more than one cause for nasaling, that of obstruction in the nose or nasopharynx, and concluding, therefore, that the nasopharynx being free, obstruction in the nose must be at fault, had found some new portion of the patient's utterly unoffending nasal organ to remove until the nasal space had become quite enormous. But not the slightest change had any of the numerous operations accomplished towards improvement of the voice. What had occurred was this: Earlier in life adenoids had so nearly taken the place of a closed soft palate that the velum had ceased to respond for the nasal sounds differently from its response for the oral sounds; thus it had remained during speech constantly raised against the posterior wall of the pharynx, not only for the oral but also for the nasal sounds, instead of dropping when a nasal sound came into the speech; and through the inculcation of habit this had gone on just the same after the adenoids had been removed. It was, then, a case of rhinolalia clausa functionalis. In Gutzmann's case two weeks of training served to entirely correct the nasaling for which the nose had been repeatedly and unnecessarily mutilated. Similar badly advised operations are not very uncommon, and the only way to prevent them is to be fortified with a knowledge of the etiology and diagnosis of the different forms of nasaling.

---

*Von den verschiedenen Formen des Näselens. Berlin, 1891.

The other functional form of nasaling, rhinolalia aperta functionalis, is more common following adenoid operations than the form just cited. Owing to the practically complete obstruction of the air through the nose by reason of a large adenoid mass, the soft palate fails to raise for the oral sounds, but remains continuously in a dropped position. In certain cases the elevation is physically prevented by the low position of a part of the adenoid tissue. After the adenoids have been removed the palate still adheres to its habit of inaction, the oral sounds are spoken with a nasal quality, and diagnostic tests reveal the rhinolalia aperta. The incorrect action of the velum will continue and will tend to become more and more confirmed until the habit is corrected by training. A similar and well known effect, temporary, however, in duration, results when the palate has been roughly handled during the adenoid operation. The correction of functional rhinolalia of either form is much easier if carried out as soon as possible after discovery, rather than after the habit has become thoroughly confirmed by continuation for a long period of years. The establishment of a rule with rhinologists of examining their adenoid patients a few weeks after operation, having the speech especially in mind, would be a saving adjunct to this procedure.

If the palatal paresis following diphtheria continues through a number of weeks the same "habit paralysis" of the velum will be in danger of inculcation. For this reason Gutzmann advises faradization and massage of the palate, if the nasaling following diphtheria continues longer than two or three weeks. Other causes for rhinolalia aperta functionalis are found in the childhood imitation of the speech of cleft palate sufferers, and of other persons with a nasal voice, and also occasional cases of open nasaling due to habit, for which no explanation is discoverable.

The subject of the speech in cleft palate is exceedingly complex and exceedingly important, and is only to be touched upon in a paper of this character; but there are certain facts relating to the speech in every case of organic rhinolalia mixta which the rhinologist should always bear in mind. Since in cleft palate the nose is almost always obstructed, either by a deformed septum or enlarged turbinate bodies, or the nasopharynx is occluded by adenoids, cleft palate serves as a typical example of rhinolalia mixta. While the predominant nasaling of cleft palate is usually that of rhinolalia aperta due to imperfect closure of the palate, this is quite regularly combined with a quality of nasaling from rhinolalia clausa due to nasal or nasopharyngeal obstruction. Nature, indeed, shows a disposition to produce a vicarious palate by filling the nose with hypertrophied turbinate tissue. I remember years ago of seeing in an adult a wide palatal defect extending far forward into the hard palate, which cleft was literally completely filled with a massive lower turbinate body. The effect of this natural obturator was undoubtedly very favorable to the patient's speech. Not understanding this, however, at the time, I began to consider the most favorable means for removing the enormous turbinate body, an operation which as I know now would have done the patient much harm. Fortunately, he was wiser than I and did not return for further treatment. Formerly one could see more unoperated palatal clefts than he sees today, the rule being now to operate during the first months of life, and the practical problem of the rhinologist has to do chiefly with operated cleft palate. As to unoperated palatal cleft cases, let it be remembered that operative interference with conditions producing nasal or nasopharyngeal occlusion should be undertaken only after the closest consideration of the speech conditions involved.

But after operation for closure of the palatal cleft it usually happens either that the soft palate is too short to reach close to the posterior pharyngeal wall, or else the palate is incapable of sufficient movement to perform its natural function in speech. Consequently most cleft palate patients possess to a lesser or greater degree a permanent rhinolalia aperta voice, even after everything possible for the speech, both by operation and training, has been accomplished. In such cases nasal occlusion, as has been said, is with practical regularity present, and also adenoid tissue is common in the nasopharynx.

Moreover, in these cases there is much tendency to eustachian and middle ear inflammation, leading pretty regularly to more or less loss of hearing.

Since nasal occlusion and adenoids are associated in the mind of the rhinologist as a cause of aural inflammations, his disposition is naturally towards operative procedures upon the occluded nose and nasopharynx. It is at this point that considerations relating to the speech become important. In the first place, it is to be considered that other causes for aural inflammation exist in these cases beside those having reference to nasal or nasopharyngeal occlusion. This is proven by the fact that such aural inflammations go on after operative measures have been taken, and Gutzmann declares that they go on practically without any modification resulting from such operations. When one considers the permanent opening between the pharynx and nasopharynx with the continuous rasping effect of food and fluids, at any rate on the lower portion of the mucous membrane of the nasopharynx, one is not surprised that a tendency to middle ear inflammations exists even aside from that due to occlusive causes.

In rhinolalia mixta, whether from cleft palate or other causes, one should approach the question of operation for obstruction in the nose or nasopharynx with a great deal of care. If the rhinologist operates with the free hand usually employed, aiming to produce a normal breathing space, he will often be surprised to note the harm he has done to the voice. The work of training of months may be undone by a single operation of this kind. For in rhinolalia aperta a considerable amount of occlusion in the nose and nasopharynx improves the voice. The presence of adenoids is of the greatest advantage, especially during the early period of training. Thus one should always bear in mind that the presence of rhinolalia aperta serves in a measure to reverse the ordinary indications for nasal and nasopharyngeal operation for occlusion.

The following suggestions will serve to render the subject more concrete: First, the seeming demand for operation on the nose or nasopharynx, for the purpose of aiding in the correction of a tendency to inflammation of the ears, should be weighed very carefully against the demand of the speech for the continuance of nasopharyngeal and nasal occlusion; second, operation for the improvement of the breathing space through the nose should be extremely conservative, certainly never producing a fully normal breathing space; third, operation for the elimination of the voice qualities of rhinolalia clausa should also be undertaken with the same extreme care, for a slightly obstructed voice is much more understandable than a badly open voice. And finally, operative effort on either nose or nasopharynx should proceed not by a single large operation, but step by step, with a waiting period between, so as to be able to call a quick halt if any signs of going too far become evident. Regarding the turbinates, extremely cautious cauterization would suit the conditions better than resection. Adenoids should be removed only after the clearest evidence of demand for removal of sufficient importance to outweigh the demands of the voice, and even then a partial operation should always be given preference over complete removal.

TREATMENT.

The correction of rhinolalia clausa functionalis involves the reinstitution of the habit of dropping of the soft palate for each nasal sound. The patient's ear should be educated to appreciate the quality of nasal as compared with oral sounds. He should be directed to take deep conscious nasal inhalations and exhalations, and then to make each nasal sound slowly and deeply through the nose. The appreciation of nasal quality tells him whether the air is or is not passing out of the nose; also the vibration of the bony nasal wall may be felt; but, better still, the nasal ear diagnostic tube serves this purpose. Having learned how to produce the individual nasal sounds, he then practices slowly and distinctly each vowel and certain regularly associated consonants with each nasal sound; and then individual words slowly spoken, and finally sentences.

The treatment of rhinolalia aperta functionalis aims to reestablish the raising of the soft palate against the posterior pharyngeal wall with each oral sound. Here, too, the patient should be trained to recognize the distinction between oral and nasal sounds. To this end deep breaths through the nose and through the mouth should be taken; nasal and oral sounds should be spoken freely before the patient in order to educate his ear. And the recognition of the difference between his own oral sounds when produced correctly and nasally may be aided by holding the palate back against the posterior pharyngeal wall while oral sounds are produced. A moderately stiff flexible flat aluminum nasal applicator, so shaped as to follow the contour of the palate and with its tip wound with a soft pledget of cotton, answers the purpose admirably. The patient speaks certain selected sounds with the palate held back, and immediately repeats them without the aid of the palatal elevator, attempting to bring the sound out of the mouth rather than out of the nose.

This method serves also as a means for producing passive movement of the palate; and faradization may also be employed for this purpose. Both methods are impossible in persons with an especially sensitive mucous membrane. Active movement of the palate is brought about by the employment of selected vowel and combined vowel and consonant exercises. They should be spoken forcibly and explosively, and with an effort to direct the sound out of the mouth, and to obtain the desired oral quality. It is best at first to base these exercises upon the vowel sound Ah, which demands less perfect closure of the soft palate than the other vowels, using this sound in various consonant combinations with some success before proceeding to o, oo, aw, e and the others. Especial difficulties will require a variety of expedients. The prognosis is excellent, the length of treatment dependent upon the intelligence of the patient and the duration of the disorder.

To summarize:

1. The rhinologist is very liable to error in diagnosis, treatment and prognosis where nasaling is the disturbance in question, unless he is able to diagnose the manner of production and cause of the alteration of the voice in each particular case.

2. He should be on his guard for functional nasaling from either one of the two causes following operation for adenoids. Otherwise operations unnecessary and harmful may be done.

3. Operations on the nose or nasopharynx for nasal voice should not be undertaken until a clear understanding of the cause of the nasaling has been arrived at.

4. A defective palate, for example, when due to congenital palatal cleft, serves to in large measure reverse the ordinary indications for operation for nasal or nasopharyngeal occlusion, and unless the voice indications are carefully followed, serious and permanent injury to the speech may result from such operations.

LXXI.

CAISSON WORKERS' DEAFNESS.*

By G. W. Boot, M. D.,

Chicago.

This preliminary report of the form of occupation deafness occurring in workers under compressed air was undertaken at the suggestion of Dr. Peter Bassoe, who found in his investigations of caisson workers that a large percentage of them suffered from deafness of varying degrees.

I have examined thirteen workers under compressed air. The men who follow this class of work are mostly Irish in nationality. They are an easy-going sort of people, who make good wages and spend it as rapidly as it is earned. They are addicted to drink, as a rule. This no doubt increases the damage done to the cochlear nerve by their occupation.

The hearing tests were made oftentimes under unfavorable circumstances, being made at times in saloons, in boarding houses, and in other places where it was not possible to secure quiet for tests for the whispered voice. The whispered voice, under these circumstances, could hardly be heard more than four meters by a perfectly normal ear.

In the whisper test the numbers "24," "28," "22" were used for testing the lower pitches of the voice, and the numbers "66" and "67" for testing the higher pitches.

Bone conduction was tested with a large A Edelmann fork that vibrates normally three minutes when struck strongly.

Weber was also tested with this fork.

Rinné was tested with the c Hartmann fork of 256 vibrations. The other forks of the Hartmann set were also used. No reliance was placed on the C_2 and C_1 forks of this set, because of their richness in overtones. The low limit was determined by a set of C tuning forks giving 32, 64, 128, 256,

---

*Read before the Chicago Laryngological and Otological Society, May 27, 1913.

512, 1024 and 2048 d. v. per second. The lowest pitched fork heard is recorded as the low limit without endeavoring to determine more closely the exact limit.

A Galton-Edelmann whistle was used for testing the upper limit of hearing. The normal ear hears 25,000 vibrations, as registered by this whistle, or even a little higher.

When the caloric test was used it was by using irrigations of cold water.

The terms "blocked," "bends," "chokes," and "blind staggers," applied by the men to the various conditions under which they suffered, are probably to be interpreted as follows:

"Blocked" seems to mean a middle ear affection such as is seen in going up or down an elevator in a high office building, but of a greater degree. At times the term covers rupture of the drum membrane.

The term "bends" is applied to a painful condition of the extremities which Dr. Bassoe has found to be due to spinal cord and other lesions.

The term "chokes" is applied to a disturbance of respiration which the men refer to the neck.

"Blind staggers" appears to be a condition in which there is vertigo.

Case 1.—D. R., age fifty years. Has worked under compressed air for twenty-three years. The first time he went down he was "blocked" under thirty pounds pressure. He had nosebleed when he came out. He went under pressure very slowly because his ears were giving trouble.

Present Condition.—Right drum membrane considerably retracted, luster good, light reflex distorted, translucency about normal. Left drum membrane considerably retracted, light reflex preserved.

Functional Tests.—Whisper, right, "28" equals 1 m., "66" equals 50 m.; left, "24" equals 1.25 m., "67" equals 1 m. Bone conduction shortened fifteen seconds. Weber lateralized to the left ear. Rinné, right, slightly plus; left, slightly plus. Low limit, right, equals 64 d. v.; left, equals 64 d. v. High limit, right, equals 16,000 d. v.; left, equals 19,000 d. v. No spontaneous nystagmus.

Nose and Throat.—Right lower turbinate very large. Septum deflected to the left. Pharynx normal.

Diagnosis.—Chronic tubal tympanic catarrh. Labyrinthine deafness.

This case illustrates well what may happen to a caisson worker who has some condition predisposing to tubal occlusion. He had a deflected septum and enlarged lower turbinate, and the first time he entered the lock he was "blocked." The retracted drum membranes show that the tubal occlusion had lasted a considerable length of time.

The much shortened hearing distance for the low pitched whisper "28" and "24," and the elevation of the lower limit to 64 d. v., are evidence of a middle ear type of deafness which was no doubt intensified by the work under compressed air.

The shortened bone conduction, though common to advanced age, is doubtless a part of the labyrinthine deafness. The positive Rinné is marked evidence of labyrinthine deafness, for with a middle ear deafness of the amount present in this case there must needs be considerable labyrinthine deafness present to prevent the Rinné bcoming negative.

The lowering of the high limit of hearing from the normal of about 25,000 d. v. per second to 16,000 d. v. in the right ear, and 19,000 in the left ear, makes the diagnosis of labyrinthine deafness positive.

Case 2.—E. F., age fifty-three years; Irish; married. Twenty-four years ago patient worked under compressed air. A week after quitting work he felt a little nauseated in the evening. About 2 a. m. he became dizzy and, he thinks, totally deaf. After four months he recovered some hearing in the left ear. He is a bricklayer by occupation, but has been unable to follow this occupation since, on account of his impaired hearing. Following the attack of dizziness he stayed in bed three days. When he got up he walked like a half drunken man. He has had no other dizzy spells. He consulted a physician at the time, who said that he had a "rush of blood to the head." Some other physician told him that both ear drums were "cracked."

Present Condition.—Right drum membrane thickened, opaque, retracted, with an area of atrophy in front of the manubrium. Left drum membrane very strongly retracted, thickened and opaque.

Functional Tests.—Patient is apparently totally deaf in the right ear. He does not hear the voice, the tuning forks or the Edelmann whistle. Bone conduction, slightly prolonged. Weber lateralized to the left ear. Rinné, right, minus (heard in the left ear); left, minus. Low limit, right, none; left, 2048. High limit, left, 14,000 d. v. Both vestibular systems respond normally to the caloric test.

Diagnosis.—Chronic tubal tympanic catarrh, right and left. Total labyrinthine deafness, right. Partial labyrinthine deafness, left.

This case illustrates what very serious labyrinthine disturbance may be brought about by compressed air. Thus, he developed a lesion of his vestibular apparatus a week after quitting work. He became totally deaf in the right ear and very largely deaf in the left ear. Whatever the lesion was that caused the nausea, dizziness and staggering gait, he appears to have completely recovered from it.

Case 3.—M. D., age fifty-six years; widower. Occupation, tunnel brick worker. He began to work under compressed air twenty-two years ago. He quit such work two years ago. He has often been "blocked." Denies venereal disease. Drinks very little.

Present Condition.—Right drum membrane about normal. Left drum membrane about normal.

Functional Tests.—Whisper, right, "24" and "66" equal 2 m.; left, "24" and "66" equal 2 m. Bone conduction shortened slightly. Weber not lateralized. Rinné, right, plus; leff, plus. Low limit, right, hears 32 d. v.; left, hears 32 d. v. Hears all the C forks. High limit, right, equals 19,000; left, equals 19,000. Nose and throat practically normal. Blood pressure 125 mm.

Diagnosis.—Labyrinthine deafness.

This patient has little or no middle ear deafness. The shortened hearing distance was due to the noisy surroundings under which the tests were made. On the other hand, bone conduction is shortened slightly, and the upper limit has fallen to 19,000 d. v. This degeneration in the cochlear nerve is not due to increased blood pressure, for the blood pressure is practically normal. This case illustrates typically the results on the labyrinth of working under compressed air.

Case 4.—M. G., age twenty-seven years. Patient has worked under compressed air for the last four or five years. He has had the "bends" three or four times. He has been "blocked" four or five times. Denies venereal disease. Drinks four or five drinks of whiskey a day and four or five bottles of beer.

Present Condition.—Right drum membrane normal. Left drum membrane normal in position, but luster deadened and color reddish, with a deeply injected streak along the manubrium. Septum of nose strongly deflected to the left, so that the left naris is completely occluded. Pharynx is dry and glazed.

Functional Tests.—Whisper, right, "24," "66" equal 60 cm.: left, "24," "66" equal close to the ear. Bone conduction apparently shortened. Weber lateralized to the left ear. Rinné, right, plus; left, plus. Low limit, right, hears 32 d. v.; left, hears 32 d. v. All the other forks heard with each ear, but C_4 considerably shortened in the left ear. High limit, right, 24,000; left, 20,000.

Diagnosis.—Deflected septum: tubal occlusion. Labyrinthine deafness.

In this case the greater amount of middle ear deafness in the left ear, as shown by the diminished hearing distance ("24" equals close to the ear) and Weber lateralized in the left ear, is probably the result of an anatomic peculiarity (septum deviated to the left). It is not clear why there should be so much diminution in the hearing distance for the whispered voice, with a range of hearing in the right ear of from 32 d. v. to 24,000 d. v., and in the left ear of from 32 d. v. to 20,000 d. v. The comparatively short time he worked under compressed air probably accounts for the labyrinthine deafness being no greater.

Case 5.—A. C., age fifty-five years: married. Has been a worker under compressed air at intervals since the age of twenty-two. Complains of a buzzing noise in both ears. Was "blocked" several times twelve or fifteen years ago, and became dizzy. Denies venereal disease.

Present Condition.—Right drum membrane: Luster good, color good, position good. There is an area of atrophy in the anterior inferior quadrant. Left drum membrane normal in position and luster, but thickened and opaque. Septum deflected to the left. Left naris obstructed. Throat normal. Blood pressure 130 mm.

Functional Tests.—Whisper, right, "24" equals 3 m.; left, "24" equals 3½ m. Low limit, right, hears 32 d. v.; left, hears 32 d. v. High limit, right equals 15,000 d. v.: left, equals 12,000 d. v. Bone conduction shortened about twenty seconds.

Weber lateralized to the right ear. Rinné, right, plus, but shortened; left, plus. Caloric test gives a weakened normal reaction.

Diagnosis.—Middle ear deafness on the right side. Bilateral labyrinthine deafness. Deflected septum.

This patient has quite good hearing for the low tones. His hearing for the low pitched whisper is practically normal. The marked shortening of bone conduction points toward a labyrinthine deafness, and this is made certain by the lowered upper limit, which is 15,000 d. v. on the right side and 12,000 d. v. on the left side. He seems to have had some disturbance of the vestibular nerve, as shown by the diminished caloric reaction. The dizziness he felt some twelve or fifteen years ago may have marked the time of the occurrence of the vestibular lesion. His blood pressure of 130 mm. is not abnormal for a man of fifty-five years.

Case 6.—P. H., age forty-eight years; widower. Denies venereal disease. Does not drink. At the age of twenty-five he began working under compressed air, and this occupation he followed for five years. He has often been "blocked" and had the "bends." He had an operation on the left side of his nose, with the idea of relieving trouble with his ear. This operation was not done until he had worked under compressed air nearly five years.

Present Condition.—Both external auditory canals were found filled with wax. After this was removed the right drum membrane was found to be about normal in luster and color. Transparency clear, but with a double light reflex. The left drum membrane was slightly dimmed in luster and moderately retracted. The left naris was occluded by a deflected septum. The pharynx was about normal.

Functional Tests.—Whisper, right, "24" and "66" equal 4 m.; left, "24" and "66" equal 1 m. Bone conduction shortened about twenty seconds. Weber lateralized in the right ear. Rinné, right, plus; left, plus (not heard at all by bone conduction). Low limit, right, hears 32 d. v.; left, hears 32 d. v. High limit, right, equals 20,000 d. v.; left, equals 16,000 d. v.

Diagnosis.—Deflected septum. Tubal occlusion. Labyrinthine deafness.

In this case the middle ear deafness on the left side was no doubt largely due to the nasal trouble on that side. This also may be responsible for the greater degree of labyrinthine deafness on that side.

Case 7.—W. K., age forty-five years; Irish; single. Has worked under compressed air for twenty-two years. A part of the time he worked under a pressure of forty-eight pounds. He had "blind staggers" in 1911, lasting three hours. He had "blind staggers" also five years before that. He has had paraplegia from his work. He has been "blocked" many times, and has had the "bends" and the "chokes." He takes about five drinks of whiskey daily, and ten or twelve glasses of beer. He had gonorrhea three years ago, also soft chancre. Denies syphilis. He has had a buzzing tinnitus since a too rapid passage through the lock five years ago.

Present Condition.—Right drum membrane considerably retracted, luster dim. Left drum membrane moderately retracted, but luster fairly good. Nose and throat normal.

Functional Tests.—Whisper, right, not heard; left, hears "66" close to the ear. Conversational voice, right, "24" equals 60 cm.: left, "24" equals 1 m. Bone conduction shortened thirty seconds. Weber lateralized to the left ear. Low limit, right, hears 32 d. v.; left, hears 32 d. v. High limit, right, Edelmann whistle not heard: left, equals 12.000 d. v. He hears all the C forks with both ears up to C$_4$, which he cannot hear with the right ear, though he hears it much shortened with the left ear. Caloric reaction normal.

Diagnosis.—Labyrinthine deafness.

The "blind staggers" may have been due to a disturbance in the vestibular system, but it has been completely recovered from. The continued use of alcohol no doubt contributed to the severity of the labyrinthine degeneration. Note the marked shortening of bone conduction (thirty seconds) and the lateralization of bone conduction in the left ear, which is the better hearing ear. Note, too, that though he hears 32 d. v. there is very marked lessening of air conduction for low sounds, inasmuch as he does not hear the whispered voice at all with the right ear. Note how much the upper limit of hearing has been lowered in the right ear (between C$_3$ and C$_4$).

Case 8.—M. L., age thirty-three years: Irish. Has worked under compressed air for five or six years, under a pressure of from five to twenty-five pounds. Has often been "blocked" in the left ear. Has often had an uncomfortable sensation in the left frontal sinus. Has had a singing tinnitus in the left ear for three years.

Present Condition.—Right drum membrane about normal. Left drum membrane a little thickened, luster nearly normal. Nose, hypertrophic rhinitis on both sides.

Functional Tests.—Whisper, right, "24" and "66" equals 3 m.; left, "28" and "67" equals 15 cm. Bone conduction moderately shortened. Weber lateralized to the right (?). Low limit, right, hears 32 d. v.; left, hears 32 d. v. High limit, right, equals 25,000 left, equals 24,000.

Diagnosis.—Tubal tympanic catarrh.

This man has suffered but little from his work under compressed air. He seems to have had some predisposition to tubal occlusion on the left side.

Case 9.—T. D., age forty-six years. Now a saloon keeper. Worked under compressed air for two years at a pressure of twenty-four pounds. Was never "blocked."

Present Condition.—Right drum membrane normal. Left drum membrane strongly retracted. Had polypoid degeneration of right concha media. Has chronic rhinitis on both sides.

Functional Tests.—Bone conduction shortened ten seconds. Weber lateralized to the left ear. Low limit, right, hears 32 d. v.; left, hears 32 d. v. High limit, right, equals 26,000; left, equals 25,000.

Diagnosis.—Tubal tympanic catarrh.

This man's hearing was apparently but little affected by his working under compressed air. However, the time he worked was comparatively short.

Case 10.—J. M., age forty-two years; Irish; single. Worked under compressed air eighteen years ago for eight months at a pressure of twenty-two pounds. Was "blocked." He had bleeding from the nose and the left ear as he came out of the lock. He had severe pains shortly after he came out, and for twelve hours afterwards he was out of his head. He tried to jump out of the third story window the next night. This mental disturbance lasted for three or four days. He was dizzy, but could walk straight. He has a buzzing tinnitus in the left ear. He takes a glass of beer occasionally. Has had gonorrhea, but denies syphilis.

Present Condition.—Right drum membrane lusterless, but normal in position. Left drum membrane retracted, luster good. Throat normal. Septum deflected towards the right.

Functional Tests.—Whisper, right, "24" equals 30 cm., "66" equals 15 cm.; left, "24" and "66" equals loud and close to the ear. Bone conduction much shortened. Weber lateralized to the left ear. Low limit, right, hears 32 d. v.; left, hears 32 d. v. C_4 shortened in left ear. High limit, right, equals 20,000; left, equals 19,000. Irrigation of right ear with cold water gives rotary and horizontal nystagmus to the left. Irrigation of left ear with cold water gives rotary and horizontal nystagmus to the right, but chiefly rotary.

Diagnosis.—Tubal tympanic catarrh. Labyrinthine deafness.

The vestibular disturbance in this case must have been slight, for, although he was dizzy, he could walk straight. Since he suffered from mental disturbance, the lesion of the vestibular system may well have been central. In this case, as in others, there is a want of correlation between the hearing distance for low pitched sounds and the lower tone limit.

Case 11.—G. G., age sixty-nine years; Dane. Worked under compressed air for two or three years, usually under a pressure of twenty pounds. While working under thirty pounds pressure he had the "bends." He was "blocked" once. He has not worked under compresesd air for eighteen years.

Present Condition.—Right drum membrane slightly cloudy. Left drum membrane normal. Has rhinitis with a little crusting. His pharynx shows chronic pharyngitis.

Functional Tests.—Whisper, right, "28" and "66" equals 10 cm.; left, "24" equals 20 cm.; "67" equals 10 cm. Bone conduction shortened twenty seconds. Weber lateralized to the right ear. Rinné, right, plus; left, plus. Low limit, right, hears 64, but not 32 d. v.; left, hears 32 d. v. High limit, right, equals 19,000 d. v.; left, equals 19,000 d. v.

Diagnosis.—Chronic tubal tympanic catarrh. Labyrinthine deafness.

In this case the nasopharyngeal condition is probably the cause of the middle ear deafness present. The labyrinthine deafness may be the result of his advanced age.

Case 12.—I. H., age forty-six years; Norwegian. Worked under compressed air for two years at a pressure of from three to twenty-five pounds. He had the "bends" once while working under heavy pressure. This trouble came on three or four hours after getting out of the lock. The next day he could not work. He had to stay in bed for three days.

Present Condition.—Right drum membrane retracted, luster good, thin. Left drum membrane retracted, luster good, thin.

Functional Tests.—Whisper, right, "24" and "66" equals 4 m.; left, "24" equals 4 m.; "67" equals 3 m. Bone conduction normal. Weber lateralized to the left ear. Rinné, right, plus; left, plus. Low limit, right, hears 32 d. v.; left, hears 32 d. v. High limit, right, equals 25,000 d. v. left, equals 21,000 d. v.

Diagnosis.—Labyrinthine deafness.

This patient has not much change of a labyrinthine nature. A lowering of the high limit to 21,000 may accompany a middle ear deafness, but could hardly occur with a middle ear deafness of so slight a degree as to be manifested only by a lateralization of the Weber test.

Case 13.—P. J., age forty-five years; Irish. Worked under compressed air for seven years, at a pressure of from fourteen to thirty-two pounds. He was "blocked" several times.

Present Condition.—Right drum membrane not visible for wax. After removal of the wax it was seen to be slightly retracted, but of good luster. Left drum membrane retracted, luster good. Nose and throat about normal.

Functional Tests.—Whisper, right, "24" equals 60 cm.; "66" equals 60 cm.; left, "24" and "66" equals 3 m. Bone conduction slightly shortened. Weber lateralized to the right ear. Rinné, right, plus; left, plus. Low limit, right, hears 32 d. v.; left, hears 32 d. v. High limit, right, equals 15,000 d. v.; left, equals 16,000 d. v.

Diagnosis.—Labyrinthine deafness.

Here we have a marked labyrinthine deafness, as shown by the shortened bone conduction and lowering of the upper limit to 15,000 and 16,000 d. v., respectively. The patient's age is not sufficient to account for such a marked degeneration of the cochlear nerve as is shown by the tests.

Considering these cases as a whole, we may say that the results upon the ear of working under compressed air are comprised chiefly under three heads:

1. Tubal tympanic catarrh. This occurs principally in those who are predisposed to tubal occlusion by some peculiarity of nasal development; such as a deflected septum, hypertrophied turbinates, occlusion of the nares, etc. If a person with such malformation undertakes to work under compressed

air the symptoms due to his tubal occlusion are exaggerated and eventually become fixed.

2. Some workers under compressed air develop symptoms referable to the vestibular system, either in its central mechanism, in the course of the vestibular nerve, or in the terminal end organs. Whatever the lesion and wherever it is situated, the results are recovered from completely.

3. The most characteristic result of working under compressed air is a loss of a considerable portion of the upper range of hearing, with marked lessening of the duration of bone conduction. In a few cases the whole organ of Corti seems to have been destroyed, and that suddenly. This lesion occurred after coming out of the lock, in one case a week after. The cases of this sort can be explained on the supposition of a setting free of gases from the blood and their accumulation in the cochlear tube; or possibly a labyrinthitis was set up that destroyed the organ of Corti, or a neuritis, as Wittmack found in guinea pigs exposed to noise, poisons, etc. Whatever the explanation, the vestibular portion of the labyrinth is either more resistant to injury or else it has a greater power of recuperation. In none of the cases examined was there a failure to respond to the caloric test.

The most characteristic result of work under compressed air is a loss of the upper tone limit corresponding in type to the loss of hearing due to working in extremely noisy places—as, for instance, in boiler making shops. It appears to be a slow degeneration that, starting at the upper limit of hearing, gradually extends downwards. The amount of deafness of this type occurring in caisson workers corresponds to some extent to the time the patient has spent working under compressed air. The changes causing the loss of the upper limit of hearing do not seem to be limited to the basal coil of the cochlea, but extend throughout the whole cochlea, for there is marked shortening of bone conduction for the lower pitches, which are not lost.

The middle ear deafness that occurs in caisson workers may be prevented by removing the predisposing causes of tubal tympanic catarrh. The labyrinthine deafness that results from long continued work under compressed air is incurable. I see no way of preventing it's development except by refraining from working under compressed air.

If deafness is to be minimized in these workers they should be examined before beginning work, to be sure that the nose is in a normal condition. They should abstain from alcohol. They should be examined from time to time to see whether labyrinthine degeneration has set in, and if it has, they should be urged to change occupation before the deafness has progressed far enough to interfere with other occupations.

The sudden lesions of the labyrinth are probably not preventable by any precautions that the men could be induced to take, beyond the slow decompression advised to prevent other lesions occurring in caisson workers.

LXXII.

THE MÉNIÈRE SYMPTOM-COMPLEX—A CLINICAL REVIEW.*

By Hill Hastings, M. D.,

Los Angeles.

I wish to present to you some case records of labyrinthine affections in which the Ménière symptom-complex occurred, to discuss those of special interest and to invite your discussion of the subject. I regret that I cannot hope to bring before you any new facts, nor do I presume to attempt a classification of nonsuppurative labyrinthine affections.

The subject of nonsuppurative affections of the labyrinth has lagged somewhat in comparison with that of labyrinthitis secondary to middle ear suppuration. There are two reasons why this should be true. First, suppuration of the labyrinth has offered a field wherein the clinical findings can often be verified by operation. Secondly, labyrinthine suppuration is not infrequently the cause of death. Autopsies are, therefore, frequently obtained. On the other hand, it is an exceedingly rare event to be able to verify by autopsy one's clinical observations in nonsuppurative affections of the labyrinth, death having resulted from other causes remotely related to the ear affection. For these reasons it is incumbent upon us as clinicians to review our past clinical experience, readjust our methods of examination in accordance with recent scientific investigations, and thus do what we can to illuminate by clinical experience the still dark portion of the ear. One must often regret that the hidden nerve field of the ear cannot be seen by the otologist as is the fundus of the eye by the ophthalmologist.

I have recently had occasion to review one hundred and forty-seven of my own records of nonsuppurative cases in which the functional tests showed involvement of the auditory nerve apparatus. Seventy-four cases (about 50 per cent) were without definite middle ear signs or symptoms. The remaining seventy-three cases showed signs of labyrinthine deafness, classified as follows: In otosclerosis, thirty-seven cases; in chronic catarrhal otitis media, twenty-three cases; in tuberculosis of the ear, thirteen cases. (Tubercular cases are included because the labyrinthine destruction did not follow the usual course found secondary to middle ear abscess.)

---

*Chairman's address, Western Section American Laryngological, Rhinological and Otological Society, Oakland, California, April 15, 1913.

Of the seventy-four labyrinthine cases (not secondary to middle ear disease) twenty (27 per cent) presented Ménière's symptoms. In eight cases the dizzy spells were of the apoplectic form.

The term "Ménière's disease" has been rather loosely applied. The classical case, reported by Ménière in 1861, served to direct attention to the anatomic seat of a diseased condition that caused a characteristic combination of symptoms—deafness and dizziness, with nausea or vomiting. Ménière's interpretation of his findings in this case has been questioned. The hemorrhagic exudate, which he found in the labyrinth, could not in itself explain the cause of death. The patient, a girl of eighteen or nineteen years, died in five days, and the autopsy showed no middle ear suppuration and no suppurative intracranial lesion. Bezold believed that the case was one, not of hemorrhage in the labyrinth, but of epidemic cerebrospinal meningitis, with extension to the internal ear—a sporadic case in which the meningeal condition was overlooked. While this is the most plausible explanation of Ménière's case, the term "Ménière's disease" has been generally applied to nonsuppurative affections of the labyrinth or auditory nerve apparatus in which Ménière's symptom-complex occurs. And Ménière restricted the classification to cases in which nerve deafness and not middle ear deafness is found.

Cases presenting Ménière's symptom-complex as a rule result from (a) diseased conditions of the blood vessels supplying the labyrinth, or (b) disease of the eighth nerve, including its end organs in the labyrinth. From a therapeutic viewpoint, cases of Ménière's disease properly belong to the general practitioner or to the neurologist.

(A) Cases of the vascular class include: (1) Hemorrhagic or (2) serous exudates[16] in the labyrinth: (3) embolism of the internal auditory artery or its branches (the terminal arteries are end arteries like those of the brain[1]); (4) embolism; (5) arteriosclerosis[15] (responsible for sudden anemia and possibly for nerve atrophy); (6) aneurismal pressure on the nerve; (7) leukemia (probably responsible for hemorrhagic exudates or nerve impoverishment; (8) severe anemias.

(B) Cases due primarily to neuritis include: (1) Toxic neuritis (salvarsan, alcohol, quinin, etc.); (2) infectious neuritis (syphilis, mumps, influenza, typhoid, tuberculosis, cerebrospinal meningitis, diabetes); (3) tumors of the eighth nerve itself and tumors pressing upon the eighth nerve (cerebellopontine angle—other nerves frequently affected at the same time); (4) meningitis with adhesions (other nerves frequently affected).

CASE REPORTS.

I. A Case of Ménière's Disease (Apoplectic Form) Occurring During Childbirth.—Mrs. W., age thirty-eight years, referred from San Diego on account of deafness in the right ear. The deafness was noticed after the birth of her last child, six months ago. Had never had any deafness or other ear trouble prior to that time. The labor was difficult. On coming out from the anesthetic she noticed great dizziness, nausea and vomiting. She did not attach any special significance to the symptoms; had never had similar experience in previous labors (has three healthy children). The dizziness continued two or three days, then subsided. Deafness was noticed on lying on her left ear. My examination showed complete nerve deafness. Weber to the good ear; bone conduction decreased; absolute deafness to whisper, speech, C to C_4 forks and to Galton whistle.

The etiologic relation to the labor, in this case, and the sudden onset, would indicate a hemorrhage or embolism of the cochlear branch of the artery. A toxic neuritis would be also a possible explanation. Lecompte[14] reports a similar case occurring in childbirth. H. Knapp[10] reports a similar case after miscarriage.

II. A Case of Ménière's Disease (Apoplectic Form); Atrophy of the Cochlear Nerve; No Permanent Injury to the Vestibular Nerve.—Miss S., age twenty-seven years, referred October 29, 1912, on account of deafness in right ear. Two months ago, while making a purchase in a drug store, suddenly noticed a queer buzzing in the ear and sudden dizziness; had to "grab the counter to keep from falling"; did not lose consciousness; had to lie down; a doctor who was called said she had a "slight stroke." She was able to walk with help to a taxicab and went home; vomited in an hour, and off and on all day. After a day or two these symptoms passed off, but she noticed she was deaf. She did not have any headache or fever; the attack was not at the time of her menstrual flow, which had always been normal; her general health had been and is now excellent. Examination shows complete deafness in the right ear to acoumeter, speech, forks C (64 d. v.) to C_4, and to Galton; normal nystagmus reaction, caloric (hot and cold) and to turning.

I saw and reexamined this patient March 1, 1913, and found no changes from those above noted.

The suddenness of the attack, the complete deafness in the affected ear, without change in the vestibular reaction, and the absence of other signs or symptoms would indicate a hemorrhagic or serous exudate in the cochlea, resulting in complete atrophy of the organ of Corti; or blocking of the cochlear branch of the artery by an embolus.

III. A Case of Ménière's Disease (Apoplectic Form); Partial Deafness; No Vestibular Changes.—C. E. S., age thirty-six years, referred October 20, 1909, by Dr. Bryant, Glendale, on account of repeated attacks of dizziness and noises in the ear. His trouble dates back one year; began while walking on the street; suddenly fell backward and to the right; did not lose consciousness; was dizzy and nauseated for only a few minutes. He had a similar attack nine months ago, and another attack last night. Each attack is sudden, unheralded by any symptoms, and not accompanied by loss of consciousness. He always falls to the right. His family physician could find no diseased condition. He has never had syphilis; does not drink; general health is excellent.

Examination of ear, nose and throat is negative, except for bruised right auricle, due to last night's fall. The hearing in the left ear is slightly impaired; watch, one inch, ordinary conversation eight feet, bone conduction reduced twenty seconds (C 128 d. v.). Air conduction, c to c_4, reduced as follows: c, minus forty seconds, c_1, minus thirty seconds, c_2, minus twenty seconds, c_3, minus thirty seconds, c_4, minus fifteen seconds. The patient says all the tones in the left ear are sharp as compared to the right. November 3, 1909, patient returns; has had another attack, similar in every way to the others. The dizziness lasted not over a minute, "had to grab the counter in the store to keep from falling—felt as if someone had bowled me over."

April, 1913, Dr. Bryant reports that the patient is still living and in excellent health; has moved up north, and has had only three attacks in the last two years. He states that the patient had a malarial history.

The cause in this case is, of course, obscure. The symptoms are suggestive of nerve irritation, from pressure periodically applied. The fact that the patient always fell to the right (the side of normal hearing) is contrary to my own experience in these cases. They have almost always fallen or had a tendency to fall to the diseased side.

IV. A Case of Mild Form of Ménière's Disease—Probably Tobacco Neuritis.—W. F. S., age forty-eight years, referred by Dr. Ross Reed, Pasadena, July 3, 1912, on account of sudden deafness and fullness in right ear and dizziness Trouble dates back one month; thought it was probably due to sudden blast from an automobile horn; has never had previous ear trouble; has not had a cold in the head; no catarrhal symptoms. General health good; denies syphilis; is married; has several children; does not drink, but has always smoked excessively, cigars and pipe; stopped smoking ten days ago.

The dizziness comes on at times; the spells are not severe enough to fall, but make him stagger; thinks the dizziness only comes when he moves his head; "the room seems to turn around."

Examination of the ear showed no middle ear signs. Hearing in right ear considerably reduced—acoumeter, ten inches; whisper ("four" and "six"), six inches and eighteen inches, respectively; conversation, twelve inches (dim); bone conduction reduced fifteen seconds; Gelle positive; air conduction, forks C to c_1 greatly reduced; c_2 and c_3 barely heard at all; c_4 poor; Galton whistle, questionable (hears with left ear); vestibular reaction to cold water and to turning is normal both sides. Examinaion of the eyes (Dr. L. W. Mansur) was negative. After five months' treatment this patient improved somewhat. His last record (December 27, 1912) shows marked improvement in the hearing for forks, and moderate improvement to conversation—whisper, six feet; conversation, seven feet; forks, c, poor (barely heard); C, minus twenty-five seconds; c_2, minus twenty seconds; c_3, normal; c_4, normal. He had stopped smoking and had lived an easy life. He took about four ounces of potassium iodid, in five grain doses three times a day. I never could see the slightest improvement after catheterization of the tubes; in fact, inflation seemed to make him temporarily more deaf.

V. A Case of Nerve Deafness (Both Ears) Following an Attack of "Grippe"; Dizziness and Vomiting.—Mrs. W., age eighty-one years, referred October 23, 1912, on account of deafness. Two years ago patient had a severe attack of "grippe"—confined to her bed for five months; no meningeal symptoms; towards the end of the illness had severe vomiting spells with dizziness, attributed to the stomach; remembers that there were some noises in the ear; did not have earache nor ear discharge; became profoundly deaf. When she was able to get out of bed, noticed she could not walk in a straight line. Examination showed both drum membranes normal. Hearing to acoumeter, whisper and speech is gone; bone conduction (c 128 d. v.) reduced; forks C to c_4, negative in the right ear; in the left ear, could hear c_3 fork alone (thirty seconds); Galton not heard in either ear; vestibular reaction not tested.

J. M. Downey[17] reports a case of labyrinthine deafness due to "grippe," in which, in addition to the cochlear nerve atrophy, there was loss of vestibular reaction.

VI. Apoplectic Form of Ménière's Disease; Complete Deafness.—Mrs. A. S., age thirty-three years, referred April 21, 1910, by Dr. Pallette. Deafness in right ear dates back fifteen years. There was no earache and no discharge; remembers considerable dizziness and noises in the ear and that the hearing was suddenly lost and has never changed. For about ten years has had attacks of sudden dizziness, nausea and vomiting, coming on suddenly—so severe has fallen several times; last attack one and one-half years ago; the attack would not last long, and there was never any unconsciousness; attributed to the stomach.

Examination.—Middle ears normal. Left, hearing normal;

absolute deafness in the right ear to speech and all forks (C to c_4); Galton questionable (hears with good ear).

In this case the persistence of the attacks of dizziness, severe and sudden, over such a long period of time is noteworthy.

VII. A Case of Sudden Deafness, Dizziness and Vomiting.—C. F., age sixty-two years, referred by Dr. Bancroft, on account of deafness (left ear) of four days' duration. The deafness came on suddenly and without apparent cause. Noticed a "feeling of fullness in the ear and got very dizzy; had to lie down; did not fall; was nauseated and vomited." Did not have a cold; was not sick. Deafness in the right ear for years.

Examination.—Left ear, drum membrane thin, translucent, good mobility; no signs of middle ear inflammation. Hearing—acoumeter, one inch; whisper, twelve inches; speech, two feet; Rinné, positive; bone conduction reduced twenty-five seconds (C 128 fork); air conduction, c reduced fifteen seconds: c_1, fair; c_2, fair; c_3, fair; c_4, almost normal; Galton heard up to 25; vestibular reaction normal in both ears.

The age in this case would indicate a vascular change in the labyrinth, due likely to arteriosclerosis, and producing sudden anemia.

VIII. Sudden and Complete Nerve Deafness; Partial Restoration; Anemia. (?)—Mr. H. S., age fifty-seven years, referred April 30, 1910, from San Diego, by Dr. Fry. Complained of buzzing in both ears and deafness. Onset, four days ago; attributed to exposure to strong east wind and to removal of a heavy beard; thinks he caught cold, but has no signs of a recent cold in the nose, and no middle ear signs. Has always had good hearing in the right ear, but some deafness in the left ear for years. His hearing tests show complete absence of hearing in right ear to acoumeter, speech, and to forks C to c_4, and to Galton. In the left ear hearing greatly reduced—acoumeter, one-half inch; whisper, five inches; speech, three feet; forks, C, minus twenty seconds; c_1, good; c_2, good; c_3, good; c_4, not heard; Galton not heard; vestibular reaction, through turning, equal on each side, twenty seconds' duration.

May 4th (four days after my first examination) I was surprised to find that there was a return of hearing in the right ear—acoumeter, negative; speech, six inches; C fork heard well; c_1, c_2 and c_3 fairly well heard, reduced about twenty seconds; c_4, not heard at all. The patient left for England. I referred him to an otologist in London. I never heard from this case. The complete deafness, sudden onset, and partial restoration of hearing, together with the age of the patient and hard radial arteries, indicated vascular disturbances in the labyrinthine vessels; possibly local anemia of the cochlea.

Richard Lake[15] reports several interesting cases of vertigo and deafness due to arteriosclerosis.

IX. A Case of Nerve Deafness; Apoplectic Form of Ménière's Disease; Ocular Paralysis.—E. P. M., age fifty-five years, a clinic patient, came to me first in November, 1904. Right ear deafness noticed three years before; came on suddenly; ear felt stuffed up and hissing noise in ear; no pain, no discharge; was working at his trade (blacksmith), but deafness was not apparently caused by any sudden noise or blow on the ear; no headaches; has been completely deaf in right ear since. Dizziness began six months ago, off and on; mild at first, but upon one occasion, fell down unconscious for a few seconds; got up and was ashamed for fear of being thought to be drunk (patient never drinks, not a glass of beer in two months on an average). In a week or two had another attack, preceded by a trembling; since then has had three or four attacks, but never quite lost consciousness. Last attack yesterday, preceded by increase in dizziness, but no other prodromic symptoms. Double vision, first noticed blurring and overlapping of images about seventeen years ago; gradually got worse, and for past five years has been severe; has to use a ground glass on left eye to enable to walk (consulted Dr. Pardee, San Francisco, who told him the nerve in the left eye was affected).

Previous History.—No illness since childhood; denies syphilis; general health is good; is married, has seven children.

Examination.—Middle ear condition is practically normal both sides. Hearing, complete deafness in right ear for all sounds.

In this case we have a record of ocular paralysis of seventeen years' duration, while the eighth nerve became affected only three years ago.

It has been my privilege recently to see at autopsy a brain from a case likely of this kind. The patient, a woman, age fifty years, had suffered for years with disturbances of the third, fifth and sixth nerves. The vessels at the base of the brain showed multiple aneurismal dilatations and marked atheromatous changes. Death had resulted from rupture of one of the aneurisms. The brain itself was normal. This patient had suffered from ocular paralysis for years. Diagnosis made only at autopsy.

X. A Case of Deafness and Dizziness, Due to "Nervous Prostration"—Deafness Almost Complete.—Mrs. B., age fifty-two years, came to my office October, 1912. Almost two years ago had a vomiting spell—deafness dates from that time. After this spell had "nervous prostration"—had been very nervous ever since; the last two months before coming to me patient had been getting better. After this spell there were many noises in the right ear—"severe noises." Following this attack was sick in bed six weeks; was dizzy and had a tendency to fall to the right. Had some slight confusion in memory. Had no difficulty in swallowing.

Examination.—Right ear drum dull and slightly retracted, but not sufficient to account for degree of deafness. Left ear—good glistening, large calcareous deposit below, but otherwise normal. Throat in good condition. Tube opened freely and drum membrane vibrated freely, if anything a little stiff. Hearing—right, absolutely destroyed; left, normal. It is noteworthy in these cases of unilateral nerve involvement, with vestibular irritation, that the tendency is to fall toward the affected side.

XI. A Case of Ménière's Disease (Apoplectic Form); Cochlear Nerve Atrophy.—Mrs. H., age fifty years, referred March 21, 1913, by Dr. A. L. Macleish, on account of deafness in left ear. Ear trouble dates back five weeks; began with dizziness, nausea and vomiting; attributed to the stomach; was so dizzy she had to go to bed; whenever she raised up would fall forward and to the left. Dizziness and nausea lasted three days. No earache, no discharge, no fever. One year ago, after "grippe," had some stuffiness in the same ear, relieved by two or three inflations. Is positive her hearing was acute up to this sudden attack, five weeks ago. Patient has been out of bed the past two weeks; is weak and uncertain in her gait, but the dizziness has gone. Examination of the eyes (by Dr. A. L. Macleish) shows glaucoma, but no ocular paralysis. Blood examination (by Dr. Walter Brem) is negative; Wassermann reaction is negative. Aural examination—Left drum membrane practically normal; completely deaf to acoumeter, whisper, conversation, and to all forks, C to c_4, and to Galton. Caloric reaction (left) quickly developed (cold water, 66 degrees, one-half quart in left ear as against one quart in right ear); reaction nystagmus well marked, lasted thirty seconds, and accompanied by considerable nausea.

It was impossible to explain the etiology in this case. Syphilis and leukemia were excluded. The patient left for her home in the East, referred to her former consultant, Dr. Charles H. Beard, of Chicago.

Spaulding[16] has recently reported a similar case from supposed labyrinthine effusion, similar to iritis serosa or episcleritis.

XII. A Case of Nerve Deafness Associated With "Nervous Prostration"; Dizziness.—Mr. M. E. J., age fifty-two years, referred January 20, 1913, by Dr. Robert Lewis, Jr., New York. Has come to California for his health. No organic disease has been found, but has had a general nervous breakdown from long period of work as railroad man. Denies syphilis. Wassermann reaction (taken at request of Dr. Lewis) was negative. Deafness has been marked of late; rapidly grown worse in the right ear; almost complete deafness in the left ear. Has frequent dizzy spells, attributed to constipation; thinks he always tends to fall backward during the spells; "room seems to be turning around." Never any headache.

Examination.—Left membrane somewhat retracted, otherwise normal; right membrane slightly retracted. Hearing, right ear: acoumeter, one inch; whisper, three feet; speech, two feet (dim); Rinné, positive; forks C to c_4, reduced twenty-five to forty seconds; Galton heard up to 45. Hearing, left ear: absolute deafness to whisper, conversation and all forks (C to c_4) except c_3, which is faintly heard; Galton heard up to 40. Vestibular reaction is normal and equal both sides.

XIII. A Case of Atrophy of Both Cochlear Nerves; History of Hemorrhages and Dizziness.—Mrs. F. R., age fifty-two years, referred from Pomona, December 19, 1910, on account of deafness both ears. Right ear had been deaf for five years. Left ear suddenly became deaf one month ago. At present unable to hear conversation in either ear. Right side deafness began five years ago with a severe attack of dizziness and general exhaustion. Was confined to her bed for six weeks. Vomited blood and had hemorrhages from the bowels off and on for two or three months, considerable prostration. (Unable to secure any opinion as to the nature of the disease.) From this attack right ear was rendered absolutely deaf, but could hear well with left ear until one month ago. Has had no earache or ear discharge, and nothing to account for the sudden deafness. Dizziness during the first attack was so severe had to sit down to keep from falling.

Examination.—Right drum membrane dull, slight retraction, otherwise negative. Left, the same. Hearing entirely gone in right ear, to watch, acoumeter, whisper, speech, and all forks; bone concduction (c 128) reduced to twenty seconds. Hearing left ear also entirely destroyed, except for the c 128 fork, which is heard about twenty seconds. Galton not heard in either ear. Pupils react normally to light and accommodation. No paralysis of the fourth, fifth, sixth or seventh nerve. Ears had been inflated for a month without any improvement in the hearing. Tube was open and dry, and no middle ear signs to account for the deafness. Patient was married and had several children; was not questioned as to specific history.

The peculiar attack, in which there was hemorrhage from the bowels and vomiting of blood for two or three months, might have a bearing on the etiology of this case. Patient was treated in another part of the country. If a careful diagnosis had been made at the time of the hemorrhage the cause for the nerve atrophy might have been found.

ETIOLOGY.

As to neuritis of the auditory nerve, some interesting facts are established. Neuritis probably occurs more frequently than one would think, and is more easily overlooked. Wittmaack[1] states that "the cochlear branch with its associated ganglion is imbedded in an unyielding bony capsule, partly surrounded by broad lymphatic spaces"; that "the blood vessels are end arteries; that the ganglion cells are smaller than all other ganglion cells in the vertebrates; that, moreover, they are bipolar and possess medullary membranes; that these anatomic relations show the elective vulnerability of the cochlear nerve."

Paul Manasse[2] in thirty-one cases of eighth nerve deafness found that the cochlear branch alone was affected in twenty-four cases. Wittmaack states: "It is not strange that a particular group of nerve fibers should be affected. This can be observed in other nerves, i. e., the recurrent laryngeal nerve, also the optic nerve—as in tobacco amblyopia; also in neuritis from lead poisoning." So with eighth nerve disease the cochlear branch alone is often affected.

Wittmaack states: "It is presumable that in the labyrinth pathologic processes generally occur in the form of small morbid foci, in the form of small hemorrhages, or inflammatory exudates, or infiltrates, situated within the delicate membranous labyrinth. From many of the autopsy reports of cases diagnosed "Ménière's" it seems to me that a serous exudate or a sudden disturbance in the lymphatic circulation may have produced the symptoms." (Case XI may be explained in this way.) "Arteriosclerosis may produce nerve atrophy through disturbance in the nutrition of the nerve. Atrophy and neuritis cannot be differentiated clinically."

One fact stands out prominently, namely, that while optic neuritis and atrophy can be diagnosed objectively, auditory neuritis and atrophy can only be diagnosed during the life of the patient by testing the cochlear and vestibular nerves. That the clinical findings in such cases point to underlying conditions of grave concern to the patient should stimulate us to examine our ear cases most carefully.

Reports in the last year or two of neuritis occurring in syphilitics after salvarsan injections, show the auditory nerve affected about twice as often as the optic nerve. As to whether the syphilitic virus or the salvarsan is responsible for the neuritis cases, it is worth mentioning that Prof. Alexander, in six years prior to the advent of salvarsan, observed sixty-eight cases of lues of the ear; in only twelve cases symptoms appeared in the early stage (third to sixth month); whereas Prof. Finger in six months had as many cases after salvarsan treatment. Alexander, therefore, concluded that there must be an etiologic relation in eighth nerve neuritis to salvarsan injections.

A large number of reports are being made of neurorecurrence after salvarsan injections. For example, Duel (Boston meeting, International Otological Congress, August, 1912) stated that he had seen three cases lately of eighth nerve neuritis after salvarsan injections—all within the first two months—an occurrence that he had not met with in an experience of fifteen years. He mentions that Benario collected fourteen thousand cases treated by salvarsan, in which one hundred and twenty-six cases of neurorecurrence occurred. Of the one hundred and twenty-six cases, fifty-one showed eighth nerve involvement.

THE THERAPY OF MÉNIÈRE'S DISEASE.

The therapeutic measures advocated in cases of Ménière's disease have been many and of remarkable variety.

Parry[5] [9] advocated the use of a seton placed in a fold of tissues of the neck at the base of the skull.

Blake and Putnam[5] [6] [7], following Babinsky, used lumbar puncture with improvement in some cases. Dundas Grant,[4] Bradley,[11] and others, following Charcot, advised use of quinin in Ménière's disease. The use of pilocarpin, potassium iodid, complete body rest, depletion by sweating, and cathartics, is rational and advised by many authorities. The use of vaccines and bacterins, as reported by one observer (Sherman[8]), seems irrational, and his results not conclusive.

Where the labyrinth tests show complete nerve deafness of long duration, atrophy of the nerve has taken place, and treatment would, of course, be of no value in clearing up the condition; however, as the other cochlear nerve occasionally becomes affected, it is wise to attempt to ward off trouble by using those therapeutic measures applicable to the particular case. In acute cases during the course of syphilis specific treatment is, of course, to be instituted. As to whether or not salvarsan should be used is too large a question to be discussed in this paper. It seems to me that the answer to the question largely depends upon whether the threatened damage from the specific infection is great enough to warrant running the possible risk of the toxic damage from salvarsan.

In an acute case of Ménière's disease, not specific in origin, a search should be made for the underlying cause, such as arteriosclerosis or endocarditis (from which a vegetation might have been broken off into the blood stream and lodged in the labyrinthine artery); or for some of the diseased blood conditions, as pernicious anemia, leukemia, etc.; or diabetes, nephritis, etc. As the treatment depends largely on treating the underlying condition, the otologist in most cases likely would best serve the patient by referring him to a good internist or neurologist, being content with pointing out the nerve change that his examination disclosed.

REFERENCES.

1. Wittmaack: On Vertigo and Disturbances of Equilibrium in Nonsuppurative Diseases of the Internal Ear. Arch. Otol., New York, 1907, xxxvl, 461-476.
2. Manasse: Arch. Otol., October, 1907.
3. Parry, T. W.: On the Treatment of Ménière's Disease and Ménière's Symptoms by Seton. Brit. Med. Journ., 1907, ii, 83.
4. Grant, D.: Two Cases of Aural Vertigo Treated by Small Doses of Quinin. Polyclin., London, 1905, ix, 160.
5. Putnam, J. J.: The Value of Lumbar Puncture in the Treatment of Aural Vertigo. Tr. Amer. Laryng., Rhin. and Otol. Soc., 1911, xvii, 317; also Bost. M. and S. Journ., 1911, clxv, 472.
6. Blake, C. J.: Considerations of the Mechanisms of Pressure in the Production of Vertigo and Report of Cases. Bost. M. and S. Journ., 1911, clxv, 469.
7. Putnam, J. J., and Blake, C. J.: The Babinsky Treatment of Aural Vertigo by Lumbar Puncture. Journ. Nerv. and Ment. Dis., 1911, xxxviii, 540-547.
8. Sherman, G. H.: Some Experience with Bacterins in the Treatment of Aural Vertigo. Am. Med., 1912, vii, 431.
9. Parry, T. W.: Case of Paroxysmal Labyrinthine Vertigo Associated with Special Ocular Symptoms and Alleviated by Seton. Lancet, 1904, i, 649-651.
10. Knapp, H.: Case of Apoplectic Form of Ménière's Disease After Miscarriage. Tr. Am. Otol. Soc., 1905, ix, 150.
11. Bradley, J. M.: Treatment of Ménière's Disease, with Report of a Case. Therap. Gaz., 1903, xix, 660-662.
12. Beck, O.: Syphilis, the Cause of Isolated Retrolabyrinthine Vestibular Diseases. Monatschr. f. Ohrenh., xiv, 1911, 505.
13. Beck, O.: Vertigo and Disturbances of the Equilibrium in Recent Secondary Syphilis. Laryngoscope, xxi, 1911, 1056.
14. Le Compte: Boston Med. and Surg. Journ., Vol. cliii, No. 14.
15. Lake, Richard: Lancet, December 14, 1912.
16. Spalding, James A.: Sudden, Total and Permanent Deafness in One Ear from Supposed Labyrinthine Effusion, Followed Four Years Later by Similar Conditions in the Other Ear. Annals of Otol., Rhinol. and Laryngol., December, 1912.
17. Downey, J. M.: Am. Med., October, 1911.

LXXIII.

MALIGNANT TUMORS OF THE NASOPHARYNX, WITH REPORT OF CASES.*

By A. L. Kelsey, M. D., and J. M. Brown, M. D.,

Los Angeles.

An exhaustive and scientific study of malignant tumors of the nasopharynx from the literature available presents many difficulties and is indeed probably impossible.

In the first place the nasopharynx is quite inaccessible for purposes of diagnosis and operative procedures; then the widespread belief that the removal of a portion of a tumor will hasten a fatal issue has undoubtedly deterred many from availing themselves of the help which can be had from the microscope; and finally the classification of malignant tumors presents some moot questions, and there is yet a lack of unanimity among pathologists as to what class a given tumor should be placed in.

There seems to be a strong movement to classify endothelioma distinctly by itself, but still it is, at times, called a form of sarcoma, while at others it is placed under the carcinomata. Again, certain malignant tumors present mixed histologic elements, while inflammatory tissue in connection with these tumors may prove a pitfall in diagnosis.

Certain benign growths may also undergo malignant changes, as is shown by many authoritative reports, and should be properly classed as some particular form of malignancy.

If, as is often undoubtedly the case, a sufficiently large mass is not removed for examination, the pathologist is unable to make a correct diagnosis, because the sections available do not show the malignant elements, which may exist in other parts of the tumor. (This happened in one of our cases at the first examination.)

---

*Read before a joint meeting of the Western Section of the American Oto-Rhinological and Laryngological Society and the Oto-Laryngological Section of the California Medical Society, April 5, 1913.

Early literature shows a mass of reports of nasopharyngeal growths, mostly loosely classed as polyps of some form, and is valueless to a study of the subject because of the lack of pathologic reports.

Undoubtedly a considerable number of cases have not been reported as yet. We recall several occurring in Los Angeles which are not used in this paper, but which will be reported later.

Chevalier Jackson in a paper published in 1901, dealing with carcinoma of the nasopharynx, quotes Moritz Schmidt as saying that in 32,997 nose and throat cases he had not seen a single case of primary carcinoma of the nasopharynx, and that Bosworth had not been able to gather more than six cases up to 1889, one being his own.

It is significant that Jackson, in the twelve years succeeding Bosworth's report, was able to add eight more cases, including one of his own, and that we, in a rather limited experience, are able to present one case, occurring in the last six months.

It would seem that many cases of carcinoma and other malignant tumors had been reported as having their origin in other regions, when they really may have sprung from the nasopharynx, and that many cases had been reported under misleading titles. In any case, later reports seem to show an increasing number of cases, and also a tendency to a more concise classification.

We do not feel ourselves competent at this time to approach the subject with the intention of writing a complete and scientific thesis, but will be content with presenting some facts gleaned from literature referred to by the Surgeon General's Reports, the Index Medicus, the Index of Otolaryngology, and such other papers as we have been able to find. We will present our own cases, four in number, which really gives us the one adequate excuse for this paper.

For the purposes of this study we submit the following classification of malignant tumors which may occur in the nasopharynx:

Carcinomata;—Glandular and squamous.

Sarcomata; Morphologically—Round celled; spindle celled; giant celled; mixed celled.

By origin—Fibrosarcoma; osteosarcoma; chondrosarcoma; myosarcoma.

Endotheliomata:—Lymphangio-endothelioma; hemangio-endothelioma.

From all available reported cases we deduce that these tumors occur with about the following relative frequency: Carcinoma, 30 per cent; sarcoma, 60 per cent; endothelioma, 10 per cent.

We are inclined to believe that these percentages will later be altered so that the endotheliomata will appear in greater numbrs, because the later reports undoubtedly show a much higher frequency than formerly, due to a more accurate classification which places some of the sarcomata and carcinomata properly under the head of endotheliomata.

Age.—Many authoritative reports indicate that malignant tumors occur in the various organs at a much earlier age than was formerly held.

Defour cites four cases of carcinoma, including one of the larynx, under one year of age.

Reliu, one of larynx under three years.

McKenzie, two cases under three years.

Rehn, one of larynx under three years.

Brem, one of cheek under one year.

Moritz Schmidt reports one case of the nasopharynx at fourteen, and Fauvel one case under seven. This we believe to be the youngest age in which carcinoma of this region has been noted.

We call attention to these reports of the early appearance of malignant tumors in other regions with the idea of inciting a more careful investigation of growths of the nasopharynx in the young, which might result in placing some of the so-called benign growths under a malignant classification.

Dabney, in 1912, cited a case of fibroma in a boy of fourteen years, which suddenly became sarcomatous. Sections of this tumor were taken at random, which showed it to be a true fibroma, but after its removal it recurred as a sarcoma. He claims that these tumors, having their period of activity during the period of development, and undergoing atrophy thereafter, or becoming malignant, show a probable common origin for these tumors and malignant growths. However, reports available for this discussion show that the various tumors occur at about the following ages:

Carcinoma	60 per cent between 40 and 60
Sarcoma	60 per cent between 10 and 30
Endothelioma	75 per cent between 20 and 40

Sex.—The male sex seem to largely predominate, especially so with endothelioma, which shows 75 per cent to occur in males.

Heredity.—All collected cases show a very small percentage giving a history of the occurrence of malignancy in the family. Of the fourteen cases of carcinoma reported by Chevalier Jackson, only one had a positive family history.

Location.—It is often difficult or impossible to determine with accuracy the actual origin of growths of the nasopharynx during life, especially when they are of considerable size, which is often the case when first seen, so that reports must often be looked upon with some doubt. Of cases gathered, when any mention is made of the origin, it would seem that they mostly spring from the vault or base of the sphenoid, while a smaller percentage occur in the lateral wall. A few cases are reported as being placed on the posterior end of the septum.

Symptoms.—Nose.—Nasal obstruction, due primarily to interference with circulation, producing turgescence of the mucous membrane, and later to the fact that the tumor closes the postnasal space. Discharge increases with the nasal turgescence, and is at first a thin mucus with no particular odor, but when the tumor becomes infected, it becomes purulent and characteristically fetid. At times it may be blood tinged. Hemorrhage is not very frequent in carcinoma or endothelioma, but fairly common in sarcoma.

Ears.—Deafness, with or without tinnitus, is present to some extent in nearly every case, and is of the eustachian tube variety. These symptoms are peculiarly apt to appear in a severe form when the tumor is situated on the lateral wall, and particularly when it has infiltrated the adjacent tissues, instead of projecting into the nasopharyngeal space, as is usually the case. One of our cases, however, showed no deafness, owing to the fact, no doubt, that the mass, though large, was rounded in form, well outlined and showed no signs of infiltration of the walls of the nasopharynx.

Pain.—Pain is present in the majority of cases early or late, and is located in the corresponding side of the head or throat. At times a certain branch of the fifth nerve is involved alone, while at other times more than one branch is irritated. In some cases the gasserian ganglion has borne the brunt of the invasion, and upon its removal has been found to be malignant. In a few cases such findings have prompted the diagnosis of primary malignancy of this ganglion, and the real origin of the tumor, which was in the nasopharynx, overlooked.

Glands.—Glandular enlargement always occurs early in carcinoma, but the enlargement may not be noticeable until rather late. In sarcoma it is not constant, and is a late development. In endothelioma it is early, and the glands become of great size. The glandular involvement is not by any means always due to malignant invasion, but quite often is a simple adenitis. It involves both deep and superficial glands.

Fixation of the lower jaw is apt to occur as a late symptom, when the tumor infiltrates the lateral wall rather than projecting into the nasopharynx, as is usually the case, and is due to involvement of the pterygoid muscle.

Retraction of the soft palate when at rest is a symptom mentioned by Trotter, due to infiltration of the levator palati muscle, and was observed in one of our cases.

Diagnosis.—Any patient showing one or more of the nasal symptoms enumerated, and particularly when coupled with enlargement of the glands, should have the nasopharynx examined at once. If a tumor or thickening is found, suspicion should be aroused, and when possible a portion of the tumor removed for a thorough examination by a pathologist. In no other way can a final and correct decision be had, and the patient is certainly entitled to have this decision given him as early in the course of the disease as possible.

Prognosis.—The prognosis is invariably bad, though two or three cases have been reported of nonrecurrence after several months, and one after three years.

Treatment.—Nonoperative.—One case is reported where the tumor disappeared after using radium, but the patient died six months later of meningitis, leading us to believe that the tumor, though apparently gone from the nasopharynx, was really progressing and had finally invaded the brain.

The injection of alcohol, formalin, adrenalin, and Coly's fluid has been tried with little success, only two cases being reported cured; one from the use of alcohol, and one by the use of formalin. Ligation of carotid artery is credited with one cured case.

It would seem that any medical treatment might be used logically in the late cases, with the expectation of reducing the size of the mass, and thus giving some relief to the patient, but not with any hope of cure.

Operative.—Many palliative and radical operations have been done by various methods, but the final results have proven very inadequate. Of the radical methods the ones most in use are the resection of the superior or inferior maxilla, splitting of the soft palate, with or without resection of the hard palate, and resecting the external nose, so that it may be pushed to one side.

Rhinologists, generally speaking, have stood against radical operations and favor conservative procedures, such as curettage, because it has been found that a large percentage of patients radically operated have not survived the operation very long.

The following cases were seen by us in our clinic in the Los Angeles Medical Department of the University of California:

Case 1. (May 6, 1907.)—Chinaman, age sixty years. He had been complaining for some months of a rather free watery discharge, without odor, and occlusion of the nose, especially the left side. Examination showed a rather soft mass in the nasopharynx, which bled slightly on touch. It seemed to be attached to the left lateral wall, and could be seen projecting somewhat into the nasal space. There was turgescence of the nasal mucous membrane of both sides. The cervical glands were slightly enlarged, with no special tenderness.

The nature of the case was not suspected at the time of the examination, and as much of the mass as could be reached was removed by the snare and curette through the nose, without much bleeding. His symptoms were greatly improved, but seen some weeks later it was found that the growth had returned, and his condition was even worse than before. A specimen was now submitted to Dr. S. T. Black, university pathologist, who reported the tumor to be a sarcoma. Died at County Hospital, November 7, 1907.

Case 2. (January 8, 1913.)—Mrs. M., age about forty-eight years. History given by patient is as follows: A subject of tonsillitis all her life, very severe during early years. Three years ago a physician punctured a small cyst situated behind the right tonsil, which contained a serous-like fluid. Two or three weeks later the tonsil became swollen and highly inflamed, and she was confined to her bed for two or three weeks from what was thought to be an infection of the punctured cyst. A year and a half ago the cervical glands of the right side began to enlarge, without tenderness or pain, but ceased to enlarge six months ago. Two months ago she felt as if she had a cold, but there was no discharge. Since that there has been continuous obstruction of the nose. At no time has she had any pain, deafness or nasal discharge.

Examination shows the following conditions: Eyes, normal. Nose, patulous; only slight increase of mucus. Both choanæ seen to be obstructed by a large mass. Tonsils both larger than normal. Many cervical glands enlarged on both sides; one on right side the size of a small hen's egg, but no tenderness present.

The finger in the nasopharynx feels a firm mass entirely filling that space, slightly movable and apparently attached by a rather broad base to the posterior wall.

Corresponding to the right choana the tumor presents an area which feels velvety, and at this site is felt a small bud-like projection which was easily detached. Near this was a membrane which also came away, giving much better breathing space.

The removed portions were submitted to Drs. W. V. Brem and A. H. Zeiler, the university pathologists, who reported it as carcinoma.

The patient is cheerful and content, and refuses any operative interference. She was seen only once since the examination, and presented no new symptoms.

Case 3. (November 26, 1912.)—N. P., age thirty-seven; male; Greek. Family history negative. Personal history: Patient says he had syphilis fifteen years ago, and was treated for it several times since then, but has never had any symptoms of the disease.

History.—For six months he has been troubled with an excessive secretion from both nares and the nasopharynx, and for the same period he has had a gradually increasing nasal obstruction. The last month he has been compelled to breathe through his mouth only, and has lost twenty pounds in weight. Deafness has been present for about three months, at first intermittent, and lately constant.

Examination.—Ears, both tympanic membranes decidedly retracted and lusterless. Nose, lower and middle turbinates turgescent, and the nasal space full of mucus. No odor. With adrenalin the parts shrink to normal size, and a grayish mass is to be seen on both sides in the nasopharynx. On palpation a mass that nearly fills the nasopharynx can be made out. The base seems attached to the under surface of the sphenoid in the position of the pharyngeal tonsil, and spreads out laterly and downward in the walls of the pharynx. The glands of the neck in the anterior triangle on both sides were enormously enlarged.

A specimen of the tumor was secured and sent to the university pathologists, Drs. W. V. Brem and A. H. Zeiler, who reported a hemangio-endothelioma. A Wassermann test showed negative results.

On October 17, 1912, under ether, the mass was thoroughly curetted away, the operation giving rise to no great amount of hemorrhage.

The patient was seen March 26, 1913, when there was considerable mucopurulent discharge having some odor. He had been working hard during the months succeeding the operation and was feeling well. Palpation showed no return of the tumor. The cervical glands were somewhat larger.

On April 7, 1913, he was seen again, when it was found that there was a return of the growth, and he showed marked cachexia and decided increase in size of the cervical glands.

Case 4. (January 28, 1911.)—B. C., age thirty-five years; male; Austrian. Family history negative. Personal history negative. No syphilis.

History.—For six months he has noticed pain in and around the right ear. At first it was intermittent or neuralgic in character and very severe at times. Latterly it has been almost constant. For four months he has noticed a gradually increasing tinnitus, but deafness was not particularly noticed.

Nasal obstruction has been present for about one month, and limited to the right side.

Examination.—Tympanic membrane on the right side is decidedly retracted and lusterless. Left side normal. Nose negative. A mass high up on the right side of the nasopharynx can be seen with the mirror. On palpation a fairly hard mass the size of a pigeon's egg, and attached to the sphenoid, was felt. A small piece was removed and sent to the university laboratory for examination. The pathologist reported sarcoma. This patient disappeared from the clinic at this stage, so the subsequent history is not known.

BIBLIOGRAPHY.

Trotter, W.: British Medical Journal, October 28, 1911.
McCaw, James F.: Laryngoscope, December, 1908.
Dabney, V.: Laryngoscope, July 1, 1912.
Jackson, C.: Jr. A. M. A., August 10, 1901.
McBurney, C.: Medical Record, February 8, 1896.
Kuh, E. J.: Medical Record, April, 1897.
Downie, W.: Glasgow Medical Journal, September, 1909.
Thompson, St. Clair: Text Book, Nose and Throat.
Schmidt, Moritz: Die Krankheiten der oberen Luftwege.
Cushing, H.: The Pituitary Body.
Thompson, J. A.: Laryngoscope, 1900.

LXXIV.

INFLAMMATION OF THE LATERAL COLUMNS OF THE PHARYNX LEADING TO ABSCESS FORMATION, WITH REPORT OF CASES.*

By Henry L. Swain, M. D.,

New Haven.

When a tissue like the lymphoid structures in the nasopharynx and throat, possessed of certain peculiar characteristics, becomes inflamed in any one of its various portions, it might reasonably be expected that certain results would follow. Or, to put it in another way, if certain kinds of inflammation were possible in any one part of Waldeyer's ring, it certainly ought logically to be possible in any other portion. Any isolated mass of lymphoid tissue could, therefore, in a general way, be expected to act, when inflamed, exactly after the fashion of the faucial tonsil, and could have, like the latter, simple acute, follicular, rheumatic, diphtheritic and phlegmonous inflammations. As knowledge became more widespread concerning what happened to the adenoid or pharyngeal tonsil, we learned to know, as years went by, that it might have acute as well as chronic inflammation. The same is true of the lingual tonsil and at a meeting of our association, in 1898, at Pittsburg, it was my pleasure and privilege to read a paper chronicling various cases of acute inflammation of the lingual tonsil which ended in abscess formation.

In recent years much has been written in the journals concerning inflammation of the lateral columns of the pharynx, and I have a presentiment that if we continue to remove root and branch from our young children all of their adenoids and faucial tonsils, there will be abundant need for us to devote increasingly more and more attention to these masses of lymphoid tissue as well as to the lingual tonsil. Hundreds of observations exist to attest to the fact that when, in years gone by, adenoids were removed in young children and the faucial tonsils were left untouched, the latter subsequently enlarged into a perfectly healthy growth, as though needed by the system, and as though trying to supply something to satisfy this lack or want. If they, the faucials, are nowadays also to be ruthlessly removed, there will, just as surely as the sun rises and sets, be enlargement of the lingual tonsil or lateral columns of the pharynx chronicled, as there has been of the faucials following the older simple adenoid operation. And if this is logical and true, we should find not only acute inflammation of these structures, but also the phlegmonous.

---

*Read at the thirty-fifth meeting of the American Laryngological Association, held in Washington, May 5th to 7th, 1913, as a part of a discussion on Phlegmons of the Upper Air Passages.

What was true way back in 1896, before the war upon the adenoid tissue was as general and as destructive as in recent years, is of course, as you all can bear me out, perfectly true of the present time. Not only have you and I been seeing cases of quinsy of the lingual tonsil from time to time, but it has been my fortune to see several within the last few months. In one of the last cases there was a great similarity noted, which was suggested in my earlier paper, to Ludwig's angina. There was here, curiously enough, enormous swelling of the submaxillary glands, inability to shut the mouth, owing to the swelling in the floor of the mouth and elevation and protrusion of the tongue. It was impossible to get the mouth open far enough to admit a finger, still less to make an examination with the mirror, and through no skill of mine the diagnosis was finally made, after the worst danger was over, when the swelling subsided, owing to rupture of the abscess into the throat. Examination of the lingual tonsil, when possible, revealed the spot of rupture to one side of the center. What was of even more importance, in a way, as illustrating the danger which the patient ran, was the observation of a subsiding swelling of the epiglottis and aryepiglottic fold which went way around to and included the arytenoids. The patient had, therefore, been in extremely threatening danger by reason of the edema of the glottis and could have readily died from suffocation, as has been frequently observed to be the case in real Ludwig's angina.

This latter possibility actually occurred only a week previously in the care of one of my ablest colleagues, who was operating to open the abscess of a genuine Ludwig's angina. He was compelled to make a brilliant emergency tracheotomy, rescuing the patient from immediate death. When respiration was restored he proceeded to find the abscess and drained it splendidly, only to lose the patient some days afterwards by sudden hemorrhage, of unknown origin, into the windpipe.

These two cases were immediately followed, in my own experience, by the last case that I have observed of abscess of the lateral column of the pharynx, the kind of phlegmon which I wish particularly to emphasize in this communication.

We have assumed more or less arbitrarily that as the lateral column of the pharynx is no mean mass of lymphoid tissue, and as it has been known to have all of the other kinds of inflammation to which lymphoid tissue is heir, we have a logical right to expect that there might also be the phlegmonous type, and I propose to submit the facts concerning certain cases I have observed to prove that such may be the case. If this is, however, as logical as I have indicated, it would seem strange that no one would have thought it worth while mentioning, unless, as I shrewdly suspect, few have believed it really to occur. Even so patient and persistent a writer and observer as Levinstein, of Berlin, who has with great detail told about all kinds of inflammation in the anginas of the lateral columns, as he calls them, fails to say anything definite about such a quinsy. I have not found it mentioned anywhere. Indeed, few of the textbooks think it is even necessary to mention the lateral column of the pharynx, probably reasoning that it would behave as any other lymphoid tissue did, and what was the use of talking about it? Moure, of Bordeaux, in his book comes as near to what I am considering as anybody whom I have happened to discover. He is very thorough in his pictures and in his descriptions of the anatomic relations of the faucial tonsil, and says that he finds a great many times the abscesses of the faucial tonsil point posteriorly.

Take this body of gentlemen present and listening to these words of mine, representing the best thought and broadest erudition of any group of men in the country in this particular line of work which we have before us, and if abscess of the lateral columns of the pharynx was of frequent occurrence it would seem as though you must have all seen it many times. I am sure you will all, if you think a moment, recall cases of quinsy where you had tried to puncture and failed, which burst in the night, and nothing showing in front, you concluded it must have perforated posteriorly. Indeed, in some instances you have been able to absolutely observe the pus coming down from behind the palate. I myself have had numerous occasions where I have been unable to get at the abscess and have contented myself with the idea that the pus had not yet formed, only to later be forced to acknowledge that it was there all the time, and that in following the routine plan of making incisions I did not get to the spot where the pus was. It is easy enough if you have a pocket of pus and it shows white under the epithelial coating to know enough to cut in there and evacuate it. And that was just how I stumbled upon my first case of abscess of the lateral column of the pharynx. And then others followed, and I now have six or seven "sure enough" cases, my last one being within a week, as I have said, in a lad of thirteen.

As the subjective and local symptoms of all of these cases were about alike. it will not be necessary now to detain you but a very few minutes to describe what happened in one case which is doubly interesting in the fact that, like quinsies of the faucial tonsils, this of the lateral column seemed to have a tendency to recur. All of my cases have been in young people. All of them have had edema (except this last case of the boy of thirteen), and the edema has, in the case of the recurrent quinsy of the lateral column, been of the most threatening type.

In the case of a young student in our university, whose history I am about to relate, there seems to be no doubt that we have had three genuine occurrences of abscess in the same situation, and I am responsible for the fact that two of them were in the lateral column of the pharynx, in the same region, and on the same side.

The patient, a young man of nineteen, had been in my charge, at our infirmary, for two weeks with an active double otitis media. One side, the left, had subsided quickly; the other, being much more active, gave us much more trouble to control. At the end of the second week he suddenly had a very sharp rise of temperature and a chill. He broke out with the most profuse erythematous eruption that I have ever seen. The body was completely covered with an urticaria, as it proved to be, and the first thought was that we had to do with a very active case of scarlet fever. With the beginning of the hives he had a very marked case of indigestion, if one chooses to use the term. The patient was nauseated, vomited and could retain nothing on his stomach for forty-eight hours. In the middle of this he began to have a sore throat. This latter symptom was, owing to the various other things which had to be done for him, somewhat neglected. It had been going on in a mild degree for two days before it assumed so active proportions that we must needs pay attention to it. It was on the left side, the side on which the ear, fortunately, seemed to be entirely through wth its acute stage. The tonsil of that side swelled and began to bulge out into the throat, being forced forward and downward toward the median line in quite the typical way for quinsy, if one did not observe carefully the situation. Being somewhat hoarse on the third day of his apparent quinsy, we discovered that he had a very marked edema of the aryepiglottic fold of that side. The arytenoid of that side was swollen as large as your thumb, a very large, baggy swelling. At the time of the discovery of the edema we twice attempted to find where the abscess was, before the fact was revealed that the tonsil was not swollen in the way which it usually is, or at least not displaced in the way in which it usually is, and that there was considerable space between the palate and the posterior pharyngeal wall. Poking forward the posterior pillar, I discovered that the lateral column of the pharynx was enormously swollen, and with a great deal of effort (for, as is usual, the mouth opened with great difficulty and one could see in only through a very small aperture) we hooked the palate forward sufficiently to discover that the upper part of the column of the pharynx was very much swollen and pushed toward the palate. Taking my cue from two previous cases of abscess in this region which I had formerly had, I made the incision around up in back of the palate into the swollen area and evacuated a large amount of pus. At this time it was learned from his parents, who had come upon the scene, that he had on a previous occasion been troubled with a sore throat where he had the utmost difficulty one night in breathing, and they feared that he would suffocate, and where relief had come by the rupture of the abscess. No observations had been made as to where the abscess formed, but from my description of the situation as it was then, the mother felt very sure that the conditions had been exactly the same in the previous attack. In any case, they had not been able to see the place where the pus came from, following its evacuation. With the subsidence of the swelling and the retreat of the edema the urticaria vanished and the patient rapidly got well. During these days of observation which immediately followed the abscess I was able to absolutely ascertain that the opening was in the substance of the lateral column.

A year and a half later I was called to the infirmary to see this same boy, reporting that he had another attack of quinsy, which had not gone on so long untreated as before, in which there was just the appearance again of edematous swelling about the arytenoid of that side. But knowing from the previous history just where to go to get at the pus pocket. within twelve hours of the onset of the severe symptoms we located and opened the abscess, and as nearly as I can tell you it was in precisely the same spot.

It is interesting to chronicle that the hives were also present in a most profuse way, and that the patient suffered with an upset digestive tract.

Commenting on this case many interesting points occur to us. That the skin could have, together with the other tissues, what amounted almost to an edema, would seem to indicate that possibly the edema of the larynx in this case was also constitutional or systemic in origin. And this leads to the statement that the whole matter of edema of the larynx would seem to need a fresh rehearsal. It would be interesting to have us all, in the next few years, observe carefully our cases of edema and to see if we could establish some theory of their existence which is less open to objection than the old venous stasis theory which has generally been held when one had a foot, or a hand, or a leg where one desired to make clear why the swelling took place.

The investigations of Fisher, of Cleveland, have led him to believe that the edema is, in a sense, a local acidosis, and the cells of the small blood vessel walls allow of the escape of fluid into the tissues which in a state of health they do not permit. No theory of pressure or damming back of the circulation could explain, to my mind at least, why a region like the aryepiglottic fold, which receives blood from an entirely different set of vessels from those which supply the tonsil and whose lymphatics evacuate very much lower into the neck, could, by any pressure or stasis which the abscess in the tonsil itself could make, be made edematous when so far away and so out of all immediate contact with the active purulent process. Hence one could in this case quite readily accept Fisher's idea that there has been brought about in some way this local acidosis, although it is still difficult to understand why the local acidosis should be found in the aryepiglottic fold and not in some other portion of the body, assuming as we do that there is no immediate direct connection in the circulation, venous or lymphatic, between the two areas.

Now I have recently, in this season, had in my own experience six cases of edema of the larynx. In two of them there was no quinsy at all. There was inflammation of the lateral column of the pharynx in both these instances, and there was in one a most marked general—what may be called rheumatic—infection, where various joints of the body were swollen and inflamed, with rapid jumps from these to other joints, and with no persistence of the symptoms in any one place for any length of time. In three other cases, in other years, mumps seemed to be the cause of the edema of the laryngeal region, which removes the cause of pressure or stasis or infection still farther away from the larynx than in any case of quinsy or inflammation of the faucial tonsils or lateral column. So that from these various cases of edema one has to look to some other cause than simple pressure, and the general acidosis of the system which had taken place in this case of lateral column phlegmon on these two occasions seems to afford a reasonable explanation of why he could have edema in any place in the body. If the system had been so disturbed as to produce this rise of temperature, erythematous urticaria, and upset digestion, it could also make the swelling in the region of the larynx. We have still to explain why it occurred first upon the side of the inflammation in the lateral column and secondly on the other side to a slighter degree.

Examination of the urine in this case showed high acidity, large indican reaction, and no albumin or sugar.

The well known curious vagaries of the socalled angioneurotic edema point all too clearly to the fact that local areas of edema can take place in almost any part of the body, even where there is no systemic or kidney condition to be looked upon as the causal agent.

The second interesting thing which we might dwell upon for a moment in this case is the fact of the recurrence on the same side, in essentially the same region, just exactly as we have so frequently observed in quinsies of the faucial tonsil.

The third point is one which anyone desiring to argue could well maintain—was this not, after all, an abscess of the tonsil which simply pointed posteriorly, and in the process of so doing caused infiltration and swelling of the lateral column of the pharynx? The only answer that I have to make to this is that I did see here the swelling and abscess so intimately in the substance of the lateral column that I do not believe it was a quinsy of the faucial tonsil. My reason for asserting that the more flatly is the fact that in these six cases of socalled abscess of the lateral column, the swelling or the displacement of the tonsil has been really different from that usually observed in true quinsy cases. To my mind. in the true quinsy case there is almost always swelling of the tonsil itself as well as displacement of it forwards and downwards toward the median line. In these marked cases of abscess of the lateral column the pillars of the palate, the tonsil, and the whole soft palate are all displaced, but there is almost no swelling of the tonsil itself, and the posterior pillar of the palate is pushed forward and away from the pharynx wall; whereas, when abscess occurs in the dead space above and laterally from the faucial tonsil, the usual peritonsillar abscess, there is almost always a pressure backward of the posterior pillar of the palate, bringing it into more immediate contact with the posterior pharyngeal wall than would be the case with the opposite side of the throat. So that if I have rightly observed the situation, I am almost sure that there is a pronounced difference in the swelling in the two instances which one can readily observe if he carefully watches the position of the posterior pillar of the palate.

Of the other cases that I have to chronicle, one was in a young lady of twenty, who had never had a quinsy to her knowledge before, which was the first case that I had observed in whom there was also edema of the glottis, and where I made three distinct attempts to discover the abscess by puncturing into the peritonsillar space in the usual situation, only to discover that I had not been observing the case properly and that the abscess formation was really back of the palate. Here it was absolutely forced upon me by seeing the abscess practically protruding around the edge of the palate when the patient gagged, and the evacuation of the abscess was followed by the usual quick relief.

Moure speaks also of abscesses occurring in the substance of the posterior pillar of the palate. This I have also seen, and it is to my mind and eye a very different situation from what I am trying to describe as existing in the substance of the lateral column.

Between the two attacks in the patient first mentioned, I saw another student with the mumps who developed a supposed quinsy, but the real abscess formation was in the lateral column of the pharynx, as in these other instances, and he had a very profound edema which was most threatening in its aspects.

The fifth case was in a young man of twenty-two, with nothing particular to distinguish it from the others, the symptoms being all the same as in the case of the young lady first observed. It was a second abscess, the previous one having been in the usual situation back of the tonsil on the opposite side.

The last case, which I have so recently seen, was in the lad of thirteen, who had never had a previous known attack of quinsy, and where the lateral column was enormously swollen at the start. His tonsils had been removed, were very small, and only a little swollen during this attack. The first puncture of the lateral column brought out no pus, and I thought I had made a mistake in my diagnosis, and on the second occasion tried to find pus in the usual region, but failed also here. In the night the abscess ruptured, and the point of evacuation was in the lateral column, out of sight of direct vision, behind the palate.

There is nothing so particularly urgent in this presentment that should necessarily claim your attention for so long a time in a meeting of this kind, only the general interest which we all take in peculiar and rare phenomena, and the hope that

I have that in having mentioned this form of abscess, not only will some case be clearer to those of you who are here, but that perhaps the profession in general may by observing a little closer make their earlier incisions more effective, and this, as sometimes in my own cases I know, may save the patient from twelve to twenty-four hours of suffering, not to mention the relief to the pressure which in some way is causing an edema of the larynx. And a word here: whereas all these cases of edema have transpired without any actual serious interference with breathing or the symptoms being what might actually be called dangerous to life, yet I cannot contemplate the existence of a large edematous swelling on one or both sides of the larynx without feeling that the patient is seriously in danger of suffocation, and as every other thing one hears of cases that have ended fatally because of profound swelling, the possibility of locating the abscess a little earlier is one which may perhaps be a life saving device. In any case, I am grateful to you for the attention that has been accorded the paper.

SOCIETY PROCEEDINGS.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of April 22, 1913.

THE PRESIDENT, DR. J. GORDON WILSON, IN THE CHAIR.

Paper: Case of Compound Follicular Odontoma of Superior Maxilla.*

BY L. W. DEAN, M. D.

DISCUSSION.

DR. M. HERZOG said he knew comparatively little about such tumors. In general terms he might say that odontoma, as a rule, originates from embryonal inclusion. According to the paper, these tumors are quite rarely reported in man. However, reference was also made to those tumors occurring in lower animals. In the horse they are comparatively common compared with the human race, and he had examined probably four to six cases in horses. The speaker was sorry that no sections had been demonstrated by the essayist, because that would have been to him the most interesting part—to see how they were composed, and whether they contained epithelial cysts and gland-like structures. He could not say very much regarding these tumors in the human.

DR. JOSEPH C. BECK said there was one fact that ought to be brought out, namely, the use of the radiogram in the diagnosis. He recently had a case of dentigerous cyst with a retained tooth that was malformed. The condition had been treated for four years for an antrum suppuration, with an opening at the last molar region. Of course, the case was treated from a different point of view by the dentist. The X-ray picture showed very clearly a double partition of the socalled antrum, and a tooth in this dentigerous cyst or cavity. Another interesting fact in this case was that during the operation, which was performed under general anesthesia, while prying the tooth loose it slipped. Three days later the tooth was removed from the nasal cavity. It had been forced through the two thin partitions into the antrum first and on into the nasal cavity.

---

*See page 1077.

Dr. Norval H. Pierce said that the division of these cases into inflammatory and embryologic cysts was very practical from a clinical viewpoint. All of the subdivisions given by the doctor could be embraced under these two heads—the embryologic and the inflammatory. The inflammatory is the socalled dentigerous cyst that we most frequently see. It is very frequently seen as a very small protuberance or cyst at the root of a tooth, when it has been removed for some cause or other, the cyst not having been suspected before. This cyst is capable of growing to an enormous extent, filling up the entire maxillary antrum.

The differential diagnosis between these cysts and empyema of the maxillary sinus is that the cysts contain cholesterin crystals.

Dr. Pierce had seen just one case of the embryologic form of cyst, and that was hardly a cyst. It occurred in a man who was brought to the Illinois Eye and Ear Infirmary, and he had a cleft reaching from just above the ala of the nose to the tear duct. In fact, the patient was sent from the eye clinic to the ear clinic, the man having gone to the eye department on account of difficulty with the tear apparatus. There was a bridge of normal bone separating the cleft above from that below, and the speaker could very easily detect two teeth just below the eye, and, running the probe down into the fistula below, which ran down underneath the gum, there was a cavity which apparently was filled with embryologic teeth. Of course, it is readily understood that by feeling with the probe one can differentiate between bone and teeth. The man only came once. He was told to come back for operation, but never appeared again. No X-ray picture was made. This is the only case that Dr. Pierce had seen of the embryologic form of dentigerous cyst.

Dr. Dean, in closing, said that no X-ray examination was made in this case because the diagnosis was plain. The sinus in the alveolus, from which the rudimentary teeth were being discharged, made a positive diagnosis. Dental surgeons have not definitely decided as to whether it is possible to have a complete absence of a tooth of the second dentition, or its embryonic structure. In New York recently they had a case in which there was apparently a congenital absence of a tooth. The patient was seen by Dr. Ottolengui, who made a radiographic examination. The X-ray picture showed most beautifully a composite odontomata. This differs from the case which he had just cited in that the rudimentary teeth were solidly cemented together by cementum. This patient was suffering no inconvenience whatever.

Atresia of the External Canal.

DR. ALFRED LEWY showed a case of atresia of the external canal. The ear began discharging at the age of eighteen and ran for several years, but was finally cured. The woman was a patient of Dr. McBean, and was first seen by him last August. At that time there was almost complete atresia. After that she was not seen until a week or two ago, when she came in with complete atresia. The patient also has Raynaud's disease and scleroderma. (Since the reporting of the case the atresia has been cured apparently by electrolysis and the insertion of Bernays' splint.

Paper: Case of Meningitis in Which the Cisterna Magna Was Drained.*

By NORVAL H. PIERCE, M. D.

Failures and Successes in Diagnosis and Surgical Intervention of Some Intracranial Diseases, Especially From the Standpoint of an Otolaryngologist, With Report of Cases.

(ABSTRACT.)

DR. JOSEPH C. BECK's report included the following conditions: (1) Sinus thrombosis; (2) meningitis; (3) extradural abscess; (4) brain abscess; (5) brain tumor; (6) hypophysis tumor; (7) intracranial hemorrhage, with and without fracture of the skull; (8) Gasserian ganglion (intractable tic douloureaux); (9) external hydrocephalus; (10) encephalocele. Of each variety he cited at least one case, discussing such points as were new and helpful in making correct diagnosis, and dwelling upon the latest form of treatment. He also gave a summary of all cases treated, with a brief resume of points helpful in brain surgery. Discussion of symptoms, pathology and other well-known matters pertaining to this subject were omitted.

---

*See page 1081.

Of thirty-eight cases of sinus thrombosis, either with or without complications, as bulb and jugular involvement, meningitis, brain abscess and general sepsis, or some other general condition, as pneumonia in nephritis, etc., twenty-six recovered following operation. Of the twelve fatal cases, ten were complicated mostly by septic pneumonia, which came practically moribund to the operating table; the two remaining cases, which were diagnosed early and apparently not complicated, died from rapidly developing meningitis. Both of these cases were of the streptococcic type of infection. His experience with meningitis was limited to seven cases, which he operated on by the method suggested by Haynes, of which six died and one recovered. Of these seven, two cases are reported in detail.

Dr. Beck has records of only fifty-one cases of meningitis, but was satisfied that he had seen again as many in consultation or in his clinical experience. Of these fifty-one cases, eighteen had spinal punctures, and septic organisms were recovered from twelve. The diagnosis of diffuse septic meningitis was made in thirty-seven cases of the fifty-one. Of these thirty-seven cases, twenty-eight came to operation either primarily as a nasal accessory sinus, mastoid, or some other local infection, or by way of the exposure of the meninges over the seat nearest the infection; and, finally, the opening of the cisterna magna of the fourteen remaining cases of meningitis, either local, septic or serous of the type. Twelve of these cases recovered without operation, and two died. This number did not include the local meningitis with brain abscess, sinus thrombosis, etc. Of the thirty-seven cases of diffuse septic meningitis, whether operated on or not, only three recovered.

Of the sixteen cases of extradural abscesses, eleven were found at the time of operating for mastoid, sinus thrombosis and frontal sinus disease. Twelve of these sixteen cases recovered after operation. Of the four fatal cases, three were operated on and became complicated by intradural abscess, meningitis and general sepsis. One case diagnosed refused operation, but a postmortem performed revealed a large extradural abscess in the cerebellar region.

Of nineteen cases of intradural abscess, two recovered. Both of these were in the temporosphenoidal area, and the operation was by way of the mastoid tegmen route. In neither case could there be any microorganism recovered from the pus of the abscess, either in smear or culture. In one case the abscess followed a rapidly destructive mastoiditis in an influenza infection, and the second in the seventh week of a scarlet fever otitis media, in a child aged three years. Of the seventeen remaining cases, ten came to operation. Six were in the cerebellar region, two frontoparietal, and two temporosphenoidal. The seven cases either refused operation or were too far advanced to be submitted to the operation.

Of eight cases of brain tumor, there was made a correct localization diagnosis in five. This number does not include gummata. The pathologic types were cyst, osteoma or exostosis, fibrosarcoma, glioma. The locations were two in the motor area, one occipital (supratentorially), one pontine cerebellar angle, one frontoparietal, one at base of frontal lobe. Five were operated on, with a mortality of 75 per cent. In not one instance did the radiogram reveal the tumor. Spinal punctures were made in seven of the eight cases, and only in one was there any increase in pressure, and in all was a negative Noguchi globulin or Nonne test present.

The cases of brain tumor which Dr. Beck observed were several cases of acromegaly, and three other cases diagnosed only at operation or postmortem. The previously cited case, erroneously diagnosed as cerebral tumor, as well as the two following supposed nasopharyngeal fibrosarcoma cases, illustrated the necessity of refining our diagnostic methods.

His experience with cases of intracranial hemorrhage had taught him that it was very easy to diagnose them, and that early operation gave most satisfactory results, both in fractures aside from the basal and spontaneous subcortical hemorrhage. The use of urotropin in large doses, as much as one hundred and fifty grains a day, had undoubtedly aided in the treatment of nasal fractures.

He cited one case of intractable tic douloureux in which the ganglion was completely removed, but the patient did not receive the expected relief.

DISCUSSION.

Dr. M. Herzog said that it was not at all astonishing that a definite diagnosis could not be made in the case reported by Dr. Beck. The postmortem clearly showed that it must have been almost impossible to locate the tumor. The most common tumors of the brain are gliomata; next in order, sarcoma and endothelioma; the last perhaps still more common than sarcoma. He has had considerable experience in the examination of glioma—probably having seen from twelve to twenty cases. He has never seen one situated as the one reported. As a rule, gliomata develop somewhere in the substance of the brain, and in spite of the fact that it is really a benign tumor, it is infiltrating in character, so it is generally not well differentiated from the brain tissues, the matrix in which it grows, but infiltrates it more or less diffusely. There are, however, some gliomata of the brain which are free tumors, in a certain sense, and those are neoplasma which arise in the ventricles, i. e., from the base or roof of the ventricle, and they project into the ventricular lumen. They are, as a rule, sitting with the broad base on the matrix. It is generally assumed that a glioma originates from an embryologic inclusion, and in some particular cases these neoplasms show an embryonic origin in their structures. The whole central nervous system, as you know, is developed in the following manner: The blastodermic disc becomes folded in, forming an open groove, which then closes and so forms the neural canal, lined by ectodermal epithelial cells, and from these, which in embryologic development have become cut off from the surface, the whole central nervous system is developed. There are quite a few gliomata which in their interior show glandular structures which are lined by epithelial ependymal cells. He has never seen any glioma which projected as a free tumor from the floor of the ventricle into the subdural space, covered by pia, until he saw this case. He supposed that it developed from an embryologic inclusion situated near the floor of the ventricle, and that for some reason or other did not, as is common, develop upwards to the cavity of the ventricle, but developed downwards, and then, in consequence of constriction, formed a slender pedicle, which was so thin that when trying to deliver the tumor at the postmortem examination it broke. The pedicle was no larger in diameter than a thin thread, and contained vessels which were continuous with the vessels in the ventricle. In turning the brain and examining the floor of the lateral ventricle, he found that the tissue was softened to the touch, and that he had torn through it, showing that the tumor had caused softening of the brain; a change similar to what is often found in dementia paralytica, and this change produced symptoms which led the neurologists to make a diagnosis of general paresis. Some consultants thought that the symptoms developed on a syphilitic basis. The Wassermann which Dr. Herzog made was negative. The examination of the cerebrospinal fluid was also negative.

The clinical symptoms could at least partially be explained by the situation of the tumor. It pressed upon the pons and the medulla, and also upon the lobe of the cerebrum. Dr. Herzog was not present at the last moments of the patient's life, but he understood that there suddenly developed a dyspnea, a condition of passive congestion over the whole surface. The heart kept on beating, but respiration stopped. The respiratory center was undoubtedly compressed upon.

On histologic examination, syphilis (gumma) could, of course, be excluded. Sarcomata sometimes spring from the choroid plexus, or from the tissues lining it, and from the pia arachnoid. These tumors also may develop almost anywhere in the brain.

The speaker made the postmortem examination about three hours after death, at night. The assistant took the brain and tumor and put it in Kaiserling No. 1 instead of formalin. Kaiserling No. 1 is unfit for preserving brain tissue for studying later microscopically to the best advantage. So he could not use special stains, such as Weigert's stain for glia fibers. The tumor, histologically, was composed of cells which were either round or oval. It was very vascular. It had comparatively large vessels, but these were not as irregular and thin-walled as often seen in a sarcoma. A very characteristic feature was that the cells were arranged around the vessels and appeared to spring from around the vessel walls. This is particularly characteristic for gliomatous neoplasms. In this tumor, in spite of the fact that he had not been able so far to demonstrate the characteristic glia fibers, by excluding all other tumors, it could be said to be a glioma of very unusual situation. It was a very unique and peculiar tumor—of unique development because it projected not into the ventricle, but to the outside of it, and only connected with its matrix by a very slender pedicle.

Dr. J. Holinger said that he had spoken at the meeting of the International Otological Association in Boston about the diagnosis of brain tumors which have caused absolutely no other symptoms except those from the ears. His attention was first drawn to this subject by a patient with brain tumor who told him that the trouble in the ear preceded by years any other symptom. That fact led him to make a diagnosis and propose operation to a patient who, eleven and a half months later, was operated on for the very thing that he had diagnosed from the functional tests so long before.

The third case that he saw was a patient with a gumma, who gave exactly the same response to the functional tests, but no other symptoms. He got well, of course, after a few injections of mercury.

The fourth patient was a lady whom he had the chance to watch for several months, who was operated on two years ago, after a prominent neurologist in this city said, only a week or two before the operation, that he did not dare make a diagnosis of brain tumor on the other evidences. Still the speaker stuck to his diagnosis, and the tumor was found, although it could not be removed. The patient is still living, and although several symptoms are developing which might suggest a second operation, the scar in the skull is still pulsating, showing that the intracranial pressure is not so very high, and therefore he would not yet insist upon another operation.

With reference to the case of Dr. Pierce, of meningitis, the speaker reported a case which was cured, in the *Zeitschrift für Ohrenheilkunde*, some time ago. The conditions in that case were in every way favorable, and he had an opportunity seven months later to verify the diagnosis at the postmortem, as the woman died from another attack of meningitis after otitis media. He could distinguish the old changes and the new ones, side by side, and could see why recovery was not possible the second time.

He has at present a case of meningitis in a young lady of seventeen, who had distinct symptoms of nasal trouble. The

X-ray showed cloudiness of the ethmoidal and frontal sinuses, on one side. That cleared up. She was up and around for several weeks. Suddenly she became unconscious and remained so for twenty-four hours, when the speaker saw her, and at once had a lumbar puncture made. The fluid was under high pressure and clear. Six or seven years previously that young lady had had a tubercular affection of the external canal and middle ear of the same side, which he had operated on, as a typical mastoid. Therefore, the possibility of a brain abscess was clear. So he suggested trephining in the temporal lobe. He made an opening in the bone the size of a half dollar. There was absolutely no pulsation of the dura. He inserted a very slender knife and found nothing but clear fluid. He explored very carefully in different directions, but while operating the pulse rose from fifty-two to sixty-five and seventy, and she clearly improved. The next morning she was clear enough mentally to recognize her surroundings. In the course of time she recovered sufficiently so that she is walking around now and doing well in every way. She does not complain of headache or any other symptom except a slight weakness in the right leg and right arm. His diagnosis in this case is that it was a case of tubercular meningitis of the base, one of the few cases that recover. Whether she will get entirely strong or not, he does not know; at present, however, she has the appearance of going to complete recovery.

Dr. John A. Cavanaugh said it has been his good fortune to have seen two or three of the cases mentioned by Dr. Beck, and what impresses him most was a complaint of headache by the patients. The other signs were practically negative. One of the cases, a young man, Dr. Cavanaugh had seen while Dr. Beck was out of the city. Spinal punctures were absolutely negative. The only thing of any importance whatever was the headache. At times the patient was a little irritable. The only thing that suggested the brain complication was the fact that a mastoid had been done, and the probabilities were that there was an involvement in the brain secondary to the operation.

Dr. Cavanaugh examined the patient and thought it best to expose the dura in the region of the mastoid and explore, but while preparations were being made for the operation the patient became cyanotic and died.

In the case of the elderly man, reported by Dr. Beck, spinal puncture just before operation was negative. The only symptom of importance was the headache.

Dr. L. W. Dean, of Iowa City, Iowa, said that the only treatment that had been of any value in his experience, so far as diffuse septic meningitis was concerned, was the prophylactic treatment. He has done a subdural drainage on some eight or nine cases. They all died. He questions the advisability of being guided by the rule laid down in Neuman's table, which states that in individuals with loss of hearing, functionating semicircular canals, no fistula symptom, and chronic otorrhea, only a radical operation, not a labyrinthectomy or labyrinthine drainage, should be performed. Following his interpretation of this rle, he did not operate upon the labyrinth in the following case. The patient had a discharging ear for fifteen years. Caloric test was positive. There was no hearing. Following the rule of the Vienna school, he did only a radical operation. Keeping in mind that the Vienna school suggested that in such cases a very careful examination should be made to see if there was any evidence on the inner wall of the tympanum of involvement of the labyrinth, he made a very careful examination. No necrosis or evidence of fistula was discovered. The head of the stapes was in good condition.

Following the radical operation, nothing of moment occurred until two weeks after, when the patient developed a slight facial paresis and a temperature of about 100.° The next day he had projectile vomiting, paresis of the facial, rigidity of the muscles of the back of the neck, temperature of 103,° and extreme headache. Spinal puncture showed a very turbid, white fluid under great pressure. It contained 97 per cent polynuclear cells. No bacteria could be found either in coverglass preparations or by making cultures on Loeffler's blood serum or agar. Fehling's solution was not reduced. Noguchi globulin test was positive. In his judgment this case was not at this time a diffuse septic meningitis, but was a circumscribed meningitis, with a general serous cerebromeningitis. A labyrinthectomy was performed. Before operation the caloric test was positive. There was no pus either in the semicircular canals or in the cochlea. As soon as the internal auditory canal was penetrated, half a thimbleful of pure white pus escaped. This was followed by two or three ounces of turbid cerebrospinal fluid. This patient made apparently a most beautiful recovery. His headaches left him. The rigidity of the muscles of the back of the neck disappeared. The temperature became normal, and he was able to sit up in bed. About ten days after the labyrinth operation, with drainage of the meningeal spaces, rigidity of the muscles of the neck became suddenly manifest again. Temperature was elevated. Spinal puncture gave the same findings as in the first case, except that in addition streptococci were found present. This case was now considered one of diffuse septic cerebromeningitis, and the condition practically hopeless. The patient died. The temporal bone is being decalcified and has not yet been examined.

If he had a similar case now he would not be guided by the Vienna school and be content with a radical mastoid operation, but would perform a labyrinthectomy.

Dr. S. A. Friedberg spoke of the difficulties in differentiating various intracranial lesions outside of sinus thrombosis or distinct meningitis. With Dr. Loeb and Dr. Beck, he had that day seen a case at the County Hospital—a boy of about twelve, with a history of suppuration of the right ear for five or six years. He had scarlet fever up to three weeks ago, from which he had made a recovery, and examination of the ear showed a central perforation—some purulent discharge—no mastoid tenderness. The boy was brought to the hospital two day before, at which time the following history was elicited: He had been having severe frontal pain for four or five days—had vomited once two days before admission, and again the night before coming to the hospital. Pulse on admission was around 64; temperature 99.° That was about the highest the temperature had gone. The speaker, with Drs. Loeb and Beck, examined him and found that he had a peculiar type of respiration—socalled air hunger, every few minutes yawning, as if getting insufficient air. Pulse still kept down; temperature not above 99.° Complained of headache. Dozed a great deal. When awakened he responded readily to questions. He spoke of having some double vision, but this had disappeared. The speaker thought there might be a slight ptosis of the right eye, but this was not definite. There was no evidence of any other involvement of the cranial cavity.

He had nystagmus—both horizontal and rotary—which was spontaneous to either side, perhaps slightly more pronounced on being directed to the opposite side. In testing with the cold water the nystagmus was produced for about twenty to twenty-five seconds. Spinal puncture showed the fluid not under pressure, clear, no bacterial content, and reduced with sugar to a certain amount. No rigidity of neck; no distinct meningitis. X-ray negative. Dizziness was complained of. In testing for ataxia, he fell to either side. The hearing, outside of the middle ear process, was pretty fair. There was one other sign elicited—diadochokinesia. On the whole, the patient seemed rather apathetic.

The speaker thought meningitis and labyrinth trouble could be excluded from the diagnosis. The question was as to the localization of the trouble. He had practically no focal symptoms. Of course, the evidence was there of pressure, as manifested by the pulse and the spinal puncture. Whether it was cerebral or cerebellar, he had not determined as yet. He simply brought this up as pointing to the difficulties encountered in localizing these intracranial conditions at times.

Dr. H. W. Loeb, of St. Louis, said that as this seemed to be in the nature of an experience meeting, he would briefly cite a case of a child, perfectly healthy except for a running ear, which had, from the history, been entirely healed for several months, who was suddenly taken with acute otitis media. The mother said that the child had put a piece of paper in his nose. This history, however, was not elicited until some four or five days after being seen. He found a large piece of paper in the right side of the nose, and was quite sure that the acute exacerbation had its inception in that. The child was taken almost immediately with symptoms which were taken to be those of meningitis—he had a positive Kernig and other symptoms that go with meningitis. Mastoid opened up and the middle fossa at the same time, but nothing was found. The symptoms continued, though somewhat modified, the spasms having been replaced by twitchings, the child going into a comatose condition. So he decided to operate in the posterior fossa. In order to get plenty of room, operation was made through the occipital bone, posterior to the mastoid wound, and not through the mastoid opening. A large opening was made, a large button taken out, incision made into the cerebellum and no abscess found, although abscess of the cerebellum had been diagnosticated. Postmortem examination showed that there was a very small abscess in the cerebellum, which had not been reached by the operation. Hence he said it was a case of successful diagnosis and an unsuccessful result, because he did not happen to strike the pus. The postmortem showed no meningitis present.

Dr. G. W. Boot reported a case, seen several years ago, of a young man who had a chronic suppurative otitis media for several years and who was taken with symptoms pointing to brain abscess. He was operated on by the speaker, who cleaned out the mastoid, opened the dura and sinus and explored the cerebellum for an abscess. This patient died after several days, and the postmortem showed that he had a cerebellar abscess, which the speaker had only missed reaching by a quarter of an inch.

He thinks we should always remember, in such cases, to look for the abscess not far from the internal auditory meatus. It is apt to be there instead of farther back.

Another case was referred to him for diagnosis of the cause of headache. In examining the young woman he found that she had very marked choked disc. He took her to see Dr. Wilder, who confirmed that, and advised him to take her to a neurologist. This was done. The neurologist thought she had a brain tumor but could not locate it., He returned the patient to the first doctor, who operated that afternoon or the next day, intending to do merely a decompressive operation. There was considerable hemorrhage at the time from the diploe. No tumor was found and the patient died the same night. The brain was removed and hardened and later sectioned, but no tumor was found. However, there was a hemorrhage occupying a space about half an inch in diameter in the cerebellum.

In this connection he thinks it is imperative to remember that the symptoms of brain tumor may be imitated by a marked anemia, and in every case where we wish to exclude the things which might be confounded with brain tumor we should always remember the possibility that the same symptoms may be set up by anemia.

Another point in connection with brain tumors is that where brain tumor is suspected, we should be very careful about lumbar puncture, because when the pressure is removed the tumor may force the medulla down towards the foramen magnum and so compress it as to cause sudden death.

He reported the case of a girl, about ten or twelve years of age, who had had a chronic suppuration from the ear for a number of years. He was called to see her because of peculiar brain symptoms, and suspected that she had a brain abscess. She was sent to the hospital for operation on the mastoid, but while preparing for operation, and before she was anesthetized, she died. In that case he always believed that the girl had an abscess which ruptured into the lateral ventricle.

DR. J. GORDON WILSON said that the paper and discussion had been suggestive as to the lines along which otology is developing. To limit otology to the temporal bone is to take a narrow view of its sphere of action. As the ophthalmologist is called to give his opinion in regard to cranial tumors, so the time would come when the otologist would be asked to assist in locating and diagnosing lesions of the brain. Already we see what has bene done by the application of the caloric and rotation tests. Further, as a considerable proportion of brain abscesses come either directly or indirectly within the field of the otologist or rhinologist, we are compelled to have at least some knowledge of cranial symptoms.

Another thing which has made this meeting most interesting is the careful and painstaking history of an obscure case of brain tumor. It is too often the case that in clinical records of brain tumors there is something essential wanting. There is no place in the body where the demand is greater for a full and careful report of symptoms, not only in the case recorded, but also for future reference. He wished to compliment Dr. Beck on the care with which this history was recorded.

In the paper of Dr. Pierce he recorded the microorganism he found. Dr. Wilson has always felt that we should be a little more particular in ear cases to ascertain the organism we are dealing with. By this recognition we can aid the prognosis and at times the treatment. Thus in time we shall get to recognize better the more dangerous organisms. The most fatal cases of meningitis he has seen have been associated with the streptococcus capsulatus.

Just one other point, in regard to the case referred to by Dr. Friedberg. Dr. Wilson has always felt that if in any case the nystagmus is directed to the diseased side, the great probability is that we are not dealing with a case of labyrinthine disease. One could imagine a case of labyrinth disease in which there is hyperexcitability of the semicircular canals, but they must be very rare.

Dr. Beck, in closing the discussion, said that the question of diagnosis by the aid of the X-ray in intracranial conditions has been a terrible disappointment to him. He has yet to find one case in which he could say that he had located a certain thing, like an abscess or tumor, by the X-ray, which subsequent operation or postmortem could show. Perhaps it was in the interpretations. Maybe in the technic. Of course, in cases of fracture, the X-ray was very useful, but in deep-seated intracranial lesions he has not been able to show its real value.

The speaker reported the case of a man with tumor at the base of the skull, in which case neurologists had made a diagnosis of hysteria. In looking up the literature he found that Cushing reports two such cases of pain, even after gasserectomy, which was performed in his case.

There was one case of encephalocele which was very interesting. He made serial sections of the temporal bone in this case. Professor Denker and others, who looked at them at the last Congress in Boston, agreed with Dr. Beck that there was an underdevelopment of the finger structures in the internal ear. So a point to consider in such cases is that, even if the encephalocele is cured, the patient's hearing will perhaps never be of much value. The investigation of such cases that have lived, either with or without operation, would add a great deal to the subject.

Regarding the case Dr. Cavanaugh spoke of, in which he said that the reaction in the spinal fluid was not the same as the normal copper reducing reaction, that was not the only symptom. The main symptom present was a purulent fluid—a fluid that contained microorganisms from the very beginning, and that was the reason the speaker operated.

Dr. Cavanaugh asked if it reduced to copper?

Dr. Beck replied that it did not. He operated because the fluid was infected. With regard to these cases, he would urge men to follow them up and try to get a postmortem, because we can certainly learn more from that than anything else. He would say openly that he had been successful in stealing his postmortems over at the Cook County Hospital, because the doctors are not supported in this by the authorities. Permission should be given for postmortems.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of May 27, 1913.

THE PRESIDENT, DR. J. GORDON WILSON, IN THE CHAIR.

Vincent's Angina.

DR. OTTO J. STEIN said that there was nothing unusual in the case of Vincent's angina which he presented in the boy, but he wanted to show the specimens correctly, side by side with the clinical case. These specimens are very profuse in the characteristic organisms—the spirillum and the fusiform bacillus, and in this case he thought he would try the inoculation of the opposite tonsil. He took some of the smear from the left tonsil, which was involved at the time, and after abrasion of the tissues of the right tonsil, the one not involved, he put this secretion underneath. Being anaerobic, the bacteria must be buried. Both tonsils are now ulcerated. (Later: Two weeks after showing this case, both tonsils were removed completely by Sluder technic, with no infection of surrounding tissue.)

In the second case the

Double Abductor Paresis

was rather unusual. He had not seen this man since a month ago, and he presented a typical abductor paresis at the time. The right cord showed an involvement of the internal tensor as well, with serrated edge and shortening of the cord, and a slight dipping inward of the arytenoid ridge, while the left cord showed nothing but involvement of the abductor up to the vocal process. The little chink was present in the back. On attempt to separate by deep inspiration, the cords made a quiver—just an effort and nothing more—there was no abduction whatsoever. Examination for some cause in the neck and chest revealed nothing physical. There was no evidence of anything, and only the X-ray demonstrated the presence of the aneurism which involved the entire arch around to the end of the third division of the arch. Dr. Stein understood, from what internists told him, that it is not unusual at all to have absolutely no physical manifestation of the aneurism for some time, while the X-ray pictures will show it always before such manifestations develop, as in this particular case, which demonstrates very nicely what the laryngologists can do sometimes in arriving at an early diagnosis.

Demonstration of the Struycken-Schaefer Monochord.

Dr. Alfred Lewy: A number of attempts have been made to construct a practical instrument for testing the hearing for higher tones. Besides the Galton whistle there are the Koenig rods, the Melde stimmplatten and the monochord. This one is the Struycken-Schaefer monochord. It was preceded by the Schulze monochord and others not so practical and less accurate. This instrument, as devised by Struycken and improved by Schaefer, consists of a metal frame 50 cm. long spanned by a piano wire on which there is a sliding block which alters by its position the length of the vibrating string. The number of vibrations, and therefore the pitch, depends, of course, upon the length, weight and tension of the string. The frame is calibrated so that the pitch is known by the position of the movable block. A scale for reading the pitch of the longitudinal vibrations is on the instrument. In the *Laryngoscope* for March, 1913, Dr. Goldstein published with his article a scale for translating the calibrations for transverse vibrations into pitch. Typographical error designates the pitch in capital instead of small case letters. By using both the transverse and longitudinal vibrations the range of the instrument is from about g^1 to beyond the high limit of human perception. As it can be used for bone as well as air conduction, it supplements very well the Bezold set of forks. It has the advantage over the Koenig rods and the Melde stimmplatten in not requiring numerous separate instruments, and over the Galton whistle in testing the bone as well as the air conduction. Besides, it is claimed by Schaefer, and so seems to my ear, that the highest tones are purer and more easily distinguished from the usual adventitious sounds.

It is best tuned by setting the block at 43 and then screwing the tension so that the transverse vibrations correspond to the **a 1 fork accompanying.** (This fork does not quite correspond with the Edelmann a^1.) There is a little calibrated weight accompanying the instrument for measuring the tension, but I have not found it practical.

For the lower tones—the transverse vibrations—the handle is rested on a table to act as a resonator (otherwise the tone is too thin), and the wire is struck by the cork hammer or rubbed with the little violin bow. The highest tones—longitudinal vibrations—are obtained by stroking the wire longitudinally with a felt pad wet with turpentine, benzol, carbon tetrachlorid or alcohol. A metal flask so equipped accompanies the instrument. The wire should not be fingered, on account of rusting. To test bone conduction the bone button is attached to the tip of the instrument and placed against the mastoid process while the tones are elicited.

While this instrument can be used for testing all tones from g^1 upward, its practical application is most important in the testing of the high tone limit, which with this instrument is d^7, 18,581 d. v. for the trained ear. For the Galton whistle it is said by Edelmann to be f^7, 22,097 d. v. In a number of patients in whom I made comparisons, the monochord and the Galton whistle seemed equally accurate in detecting slight losses of the upper tone limit. For the ordinary ear c^7, 16,554 d. v., may be considered a normal standard. Wolff, of Berlin, after testing forty patients with this instrument, came to the conclusion that characteristic for nerve deafness is a greater loss of perception for high tones by air than by bone conduction, the discrepancy in some cases being quite marked. That is, bone conduction is better than air conduction for high tones, the reveres being true for lower tones. This also occurs, he finds, in marked effusions of fluid in the middle ear or external canal. Normally, and in pure conduction deafness, except when effusions are present, the hearing by bone and air conduction for high tones is about the same. You will recall that with the tuning fork, a 1 for instance, the loss by bone conduction is the more prominent in nerve deafness. Struycken explains this by saying that the vibrations of the handle of the tuning fork are only about one one-hundredth the intensity of the tines. No doubt the pitch is also a factor.

My own investigations so far are limited to twenty-three cases; three of spongification, five nerve degeneration, five otitis media chronica, six mixed, two chronic suppuration, and two of doubtful diagnosis. Two of the cases of nerve deafness are believed to be due to tobacco. So far my findings agree with those of Wolff, i. e., bone conduction is better than air conduction for high tones in nerve deafness. Permit me to express here my opinion that the condition of the high tone limit is of value in prognosis, and that any considerable loss of hearing for high tones, except in hysteria, is of bad prognostic import, regardless of the original cause of the trouble.

My thanks are due Dr. Pierce for the opportunity of making these investigations in his clinic.

DISCUSSION.

Dr. Robert H. Sonnenschein said as to the difference in the appreciation of high tones by bone and air conduction, that he believes, as stated by physicists, and as mentioned by Dr. Lewy, very high tones are better heard by bone conduction than by air conduction, and the striking difference between the amplitude of the prongs and the stem of the fork accounts for that. If the amplitude is the same, high tones are heard equally well by both, but in the small c 4 and 5, the amplitude of vibrations of the stem of the fork is only one one-hundredth that of the prongs, which accounts for this discrepancy.

So far as he can see, with the use of the instrument demonstrated by Dr. Lewy, with which he has had only slight experience, it is very difficult in a great many cases, when you get up as high as c_6 and c_7 to get an appreciable tone constantly. Sometimes when you rub it you get a squeak here and there, but, as a rule, you cannot get a consistent tone. Of course, when you get down to small c 4 and 5, you can, but you can get that with a good tuning fork. So thus far he cannot see any very great advantage as compared with a small c_5 fork.

Dr. George E. Shambaugh said that he is coming to appreciate more and more that the testing of the upper tone limit is one of the most valuable tests that we have in diagnosis as well as prognosis. A c_4 tuning fork does not suffice for this test—except in advanced stages of labyrinth deafness, but where one gets an appreciable loss of hearing for the c_4 tuning fork, it may be dispensed with, because the other tests show the condition.

The testing of the upper tone limit is especially valuable in the study of cases of advanced long standing middle ear deafness. In many of these there begins to develop, sooner or later, a degeneration in the labyrinth—labyrinth deafness, which one cannot detect with the Weber and Rinné, or the Schwabach tests, because these tests will continue to give the characteristic reactions of middle ear deafness. The quickest way in which one can detect beginning labyrinth degenerations is by testing the upper tone limit. A recognizable defect in the upper tone limit is of decided significance prognostically.

Dr. J. Holinger said that Burkhardt-Merian, of Basle, was the first to recognize the diagnostic importance of the upper tone limit and used it systematically. He employed a whistle which he perfected in all details, calling it "Galton whistle." The original Galton whistle had simply the movable piston, and was not made for the highest sounds. Burkhardt-Merian determined the form of his mouth and its relation to the bore of the whistle. He introduced the screw and gauge on the piston. It ought to be called "Burkhardt's whistle." Some of these Galton whistles have done pretty good service. Others were not executed so carefully and accurately; therefore, the results were not uniform. Later on Edelmann knew that the upper limit of his series was the most unsatisfactory part. He tried to gauge his whistles by means of Schwendt's dust figures. But even in 1899, when Dr. Holinger got his set of tuning forks, there was a rumor that the Galton whistle was not scientifically accurate. It was said that in the highest pitch a turn of the screw of the piston means a rise in pitch of half an octave; in the lower region of the whistle only one or two tones. Therefore, the original Edelmann's whistle could not be considered a "scientific" instrument. It was improved later on. In 1909, when abroad, the speaker saw the monochord in use very extensively, but it was an entirely different instrument from the one demonstrated by Dr. Lewy. He had the impression then that it was not an instrument to be used in the office every day, because it was unhandy. The instrument shown by the essayist is carefully gauged and is much more handy. This is necessary in an instrument of that sort. Dr. Holinger has not paid very much attention to the determination of the higher tone limit in the last few years, but now that we have a workable instrument, he intends to take a greater interest in this part of the functional tests. The great many improvements that Edelmann made of late in his whistle made the whistle also a workable instrument, but still every once in a while we read criticisms from scientists who are in a position to really verify and prove their contentions.

DR. WILLIAM L. BALLENGER has had no experience with this instrument, although he has owned one for years. He did not understand just how to use it until Dr. Goldstein's article came out last March. He has had great difficulty with the Edelmann-Galton whistle. The instrument he has was made in 1903. It can be tuned in different pitches. That is, you cannot set it and then go up all the pitches by turning the main apparatus, but you must turn a tuning apparatus at the other end of the instrument, and he finds great difficulty in getting the same intensity of pitch, and hence he has felt that it was not a very useful instrument in his hands. But with this instrument we can have a constant pitch and greater accuracy. It is a simple instrument, and he hopes to make use of it in the future.

DR. LEWY, in closing the discussion, said that the practical advantage that the instrument he exhibited had over the Galton whistle is that it assists us in obtaining the bone conduction for high tones as well as the air conduction. The Galton whistle gives us only the air conduction. It may be that through this we will be able to add to our diagnostic and prognostic ability. He simply presented the instrument for what it was worth, and in the interests of the society.

Paper: The Nasal Voice With Reference to Its Bearing on The Practice of Rhinolaryngology.*

By ELMER L. KENYON, M. D.

DISCUSSION.

DR. J. HOLINGER thinks that there is another phase to the question discussed by Dr. Kenyon. Dr. Kenyon warned against the removal of adenoids under all circumstances, without regard to other organs. This is a timely suggestion. At present the speaker is treating a child whose adenoids had been removed about a year ago by a laryngologist who does not make any pretensions of treating ears. The boy has highly retracted drum membranes, is therefore hard of hearing, and has a decided defect of speech, consisting of an apparent rhinolalia. The speaker tried to study the case, and found that in pronouncing the patient's own name, Norman, the defect was most evident. He pronounces it Noonan. The consonants "r" and "m" are of very low pitch and not heard well, and therefore are not pronounced correctly by the otherwise bright boy. His rhinolalia is nothing but the consequence of lack of hearing. The ear was treated three or four times, and the mother was instructed to watch the boy. It was surprising how quickly he improved; he speaks much better now. Dr. Holinger thinks it is often not so much a question of instruction as a question of correcting the condition in the ear. That was his contention for many years as to the adenoid operation in the hands of men who do not treat the ear, and who do not pay any attention to the ear after the operation. They will overlook a retraction of the drum membrane, and the consequence is that they practically have not benefited the patient very much. Considering the experience with this boy's case, might there not be a possibility to explain rationally the case of Professor Gutzmann as that of a patient who did not hear well as a child and therefore missed the right pronunciation?

---

*See page 1110.

Dr. Joseph C. Beck was very much interested in the paper, and it reminded him of some cases that he had had of speech defects. One case he showed before the society several years ago, of rhinolalia aperta, in a case of congenital short palate, which, through training, before he had done anything, refused to respond. He subsequently injected paraffin behind the mucous membrane in the region where one would expect this palate would meet the posterior margin of the soft palate, and immediately after the injection this patient began to speak more clearly and distinctly, and the result was very satisfactory indeed. There was an interesting fact about this patient, in that was she was a young woman then of twenty-five, and did not know that she had this defect. The doctor wanted a record of her speech before he did anything for her, and so he thought of Dr. Brown, of Milwaukee, who had recorded his cases by phonograph, so he had her talk into a machine, and when she heard the record she was completely paralyzed and fainted away, for she did not know she had spoken that way.

When Dr. Kenyon spoke of educating the patients so that they know how they speak, it is perhaps by the aid of that nasal ear, and Dr. Beck thinks that perhaps in that way they may be able to hear how they speak. Dr. Patrick spoke to him once of a condition, the German name for which he has forgotten, in which the patient does not understand or cannot hear his own voice. In fact, the speaker thinks it is quite common, and that most of us are surprised at hearing our own voices in the phonograph—they sound different.

He was reminded of another case following removal of adenoids, in which the patient developed a distinct rhinolalia aperta, and he thought that he had perhaps traumatized the palate. There was perfect action in the palate. The condition did not improve for some time. He examined the patient very carefully, and found a very soft portion of the hard palate, and in taking a dental radiogram he found a marked deficiency of bone in the hard palate—a congenital deficiency. This voice defect was not present while the adenoids were present, but when he had removed the adenoids—which was not done for any other purpose than a distinct systemic condition—this speech defect occurred.

Another point: The doctor said it is difficult to examine the palate. Dr. Beck has examined the palate, and you can see the action of the palate during speech and see what condition it is in.

Dr. Alfred Lewy said that one idea that impressed him after hearing Dr. Kenyon's paper was the great neglect of the entire subject of speech defects by the medical profession in this country. Most of the treatment of speech defects is done by people who advertise to cure stammering, and use bizarre methods, such as causing the patient to sing the words or to go through certain peculiar motions, and so forth. In the public schools in Chicago some effort has been made to train special teachers in the correction of defective speech, but altogether in this country the subject has been very largely in the hands of charlatans, and he thinks it is largely the fault of the medical profession, and that we need such papers as that of Dr. Kenyon to show us the importance of the subject and the possibilities that lie therein.

Dr. Kenyon, in closing the discussion, congratulated Dr.

Holinger on his diagnosis in the patient whose hearing was impaired. It is often not an easy diagnosis to make. Many a child talks badly because he does not hear correctly. Many a child does not talk at all because he cannot hear exactly, and he may not be more than moderately deaf.

Paraffin injection in palatal cleft is, of course, a valuable procedure. He referred to it merely to point out that adenoid tissue serves exactly the same purpose as the paraffin—it helps to prevent the air from going upwards into the nasopharynx, when the impaired palate does not sufficiently perform that function.

It would seem as if the nasopharyngoscope passed through the nose into the nasopharynx might aid in diagnosis in difficult cases of imperfect palatal movement, by enabling one to watch the movements of the palate; but diagnosis is usually possible by the much more simple methods described.

So far as the patients not recognizing their own voices is concerned, that is very true. None of us know our own voices, and patients with the most apparent defects of speech—if they be children and have never been told about their incorrectness of speech—believe that they talk as well as anybody, and they have to be told in order to know that they have a defect of speech.

The Value of Skiagraphic Plates in the Study of the Diseases of the Nasal Accessory Sinuses and Mastoid Process.

DR. W. L. BALLENGER presented a number of very interesting and instructive photographs of skiagraphic plates illustrating the value of skiagraphic plates in the study of the diseases of the nasal accessory sinuses and mastoid process. He presented two plates which showed a single ethmoid cell involved in each instance, and brought out the fact that in such cases the operation for the drainage and exenteration of the diseased cell could be done with a minimum amount of surgical trauma. In one of these cases the patient had been repeatedly operated by a competent specialist, but without relieving the suppurative symptoms. Dr. Ballenger, by means of the plate, located a single ethmoid cell, on which he operated, with entire relief to the suppurative process. In the other plates showing a single cell involved it had been diagnosticated as a case of cerebrohydrorrhea because of the profuse watery discharge from the patient's nose. The plate, however, shows one cell located under the right frontal sinus to be involved. The interesting points concerning this case were the possibility of locating a single infected ethmoid cell, which was giving rise to a nasal hydrorrhea. There was no purulent discharge in this case, but in spite of this the cell was distinctly cloudy, as much so as in suppurative disease. It also cleared up the diagnosis. He also showed plates showing the osteoporotic destruction of the inner plate of the frontal bone from luetic disease. Also plates contrasting a healthy mastoid process with a diseased process.

Plates showing extensive fractures of the skull were also shown, in which the patient, while riding in an automobile, collided with a telegraph pole and was rendered unconscious for three or four days. On recovering he was found to have nystagmus to the opposite side, nausea and vomiting. On examination by Dr. Ballenger, two months later, the nystagmus, nausea, etc., had disappeared, but he still suffered from disturbance of equilibrium, and was totally deaf in the right ear. The plates show a large arch fracture extending from the outer angle of the left orbit to the inner angle of the right orbit, and a Y-shaped fracture extending through the temporal bone on a level with the cochlea and vestibular apparatus.

DISCUSSION.

Dr. R. C. Lynch, of New Orleans, said that he had been using X-ray plates in his work in New Orleans for the last six or eight months, and has found that they help very materially in every branch of the work, especially in the diagnosis and proper interpretation of sinus suppurations. One case of mastoiditis that gave him a good deal of concern, because of the very chronic character of the inflammation and of so few clinical symptoms that accompanied the condition, was really treated successfully on account of the use of the radiogram. This patient had the usual onset in an acute otitis; the drum ruptured. He was an ignorant man and neglected it for some time, and then began to suffer with headache, and the headache was the only symptom that he gave of any disturbance. His headache increased in intensity until he came to the clinic for observation or for some relief. The X-ray pictures showed the normal side to have a very extensive cellular mastoid, the cells extending well forward into the zygoma, very much higher than usual, over the roof of the tympanic cavity and far back, and especially the tip was very large and cellular. On the diseased side it showed the process to extend even into the diploe, and he was rather suspicious of a low grade osteomyelitis developing in this case in consequence, or, rather, as a result, of the extreme degree or the great amount of involvement of the bone, as shown by the X-ray. In doing the operation he extended it well beyond the limits that he would have, had he not had the X-ray picture at his side. This man recovered without any disturbance. Dr. Lynch believes that if he had not had a radiograph at his disposal, and the measurements taken carefully before operation by the radiologist, the extent of the operation would not have covered the diseased processes, and the consequence would have been that this patient would not have recovered in the very nice and proper way that he did under this treatment. So that he believes that the X-ray is very valuable. We are really beginning to look upon it now with far more favor than we ever have, and he believes that it is opening up more and more new fields for diagnosis. He had the pleasure of discussing this phase of this subject with Dr. Ingersoll, who has been doing some work on the internal ear. He feels, Dr. Lynch believes, that he can outline the semicircular canals, though rather indefinitely, by X-ray. It is not at all impossible, the speaker imagines, to have the X-ray help us materially in the diagnosis of certain conditions of the internal ear. He made that statement, not as a definite fact at all, but rather as a thought that came to him at the time, that it may be that we may devise some method in the future of helping us along this line. Certainly, if the X-ray can be of service in these conditions, it will be a source of pleasure and benefit to us.

Dr. Joseph C. Beck said that it was unfortunate that Dr. Ballenger could not have shown the plates instead of the photographs. It has been, by radiologists all over the world, a prohibitive thing to demonstrate facts by photographs, and he was partially to blame for this exhibition to-night by photographs, because he promised Dr. Ballenger that he would bring down these frames for showing the plates. So he wished to apologize.

It was certainly a pleasure to him to hear men express themselves in such a way regarding the use of the X-ray. He was and is very enthusiastic over it, but still he might say at this time that he is losing a bit of this great enthusiasm.

The fine points that Dr. Ballenger brought out, of localizing one single cell, he would take issue with, especially in a single radiogram, for what do we know by looking at one plane from one point of view how many cells are involved. With stereoscopic pictures, however, we can tell better how many cells are involved. The speaker would not like to go on record as saying he could make this diagnosis.

Dr. Lynch brought up the point of radiography of the semicircular canals. Dr. Beck has tried to do this, and in not a single instance in the living could he determine anything further than the internal auditory meatus, which he for a long time took to be the middle ear. If you examine with the X-ray and see a distinct outline of the middle ear, that is not the middle ear, but is the internal auditory meatus, and is diagnostic of tumors of the auditory nerve.

When we speak of taking X-ray pictures of these bony structures through bone that is as dense as that, it does not seem possible in the present stage of radiography. Stereoradiography of the sinuses and mastoid is the coming thing.

Another thing: To leave the interpretation of the condition that you are dealing with to a radiologist is a mistake. Leave him out entirely. Just see that he takes it correctly, and make your own interpretation of the print.

Dr. Ballenger, in closing the discussion, said that Dr. Beck was entirely mistaken about the impossibility of showing a single cell except by the stereoscopic method. If the print is taken from the corner of the head, as it were, through the orbit, and not the front view, in this way you get a perspective view of the cells back to the sphenoid, and as far forward as the frontal and anterior ethmoid. You can see the cells one by one from the front to the rear, and then, if you will take the opposite side, you will also see that same cell from the opposite side, but from a different angle, and in that way, by taking two plates of the cell, one cannot be misled; and if the cell is one that can be penetrated with a probe, if it happens to be so located, you can place it accurately, and only remove that one cell, as was done in one of the cases reported.

In the second case reported he removed two or three cells.

The point he wanted to make was simply that it is possible to do this without stereoscopic plates. In taking the pictures as suggested, one plate shows absolutely every sinus of one side of the head, and the second all the cells on the opposite side, and he knows of no possibility of being confused by this method.

Paper: Caisson Workers' Deafness: A Preliminary Report.*

By George W. Boot, M. D.

DISCUSSION.

Dr. Peter Bassoe (by invitation) said he was glad that Dr. Boot had reported this work to the society, because otologists can do a great deal toward clearing up various points about caisson disease, and also there are things in caisson disease that come as near animal experiments on a human being as anything can be, and if properly carried out at the proper time may help to clear up many points in otology.

We must bear in mind that the man who works in compressed air is subject to trouble when he goes into the compressed aid and when he comes out again. So far as ear conditions are concerned, the most common ones, such as "blocking," are brought about during compression, and the ear is the only organ affected by the state of compression. Of course, what happens when the air pressure is rapidly rising is that the air is all the time being compressed, there is more oxygen and nitrogen taken into the blood, and in order to avoid being "blocked" the middle ear must contain the same kind of air as the outside air, otherwise there will be unbearable pressure on the drum, hence every worker is taught to swallow air while going in, and if he does not do that he invariably gets "blocked." Some of those who have investigated very extensively along this line believe that primarily all of the ear troubles of caisson workers are due to this stage of the compression—that the chronic troubles that follow in the middle ear and internal ear are secondary to this disturbance that occurs during blocking. The work of Dr. Boot has shown that we do deal with a good deal of labyrinth trouble in many of these cases, and it seems to the speaker that he has shown pretty well that the stage of decompression has a good deal to do with it. Practically all of the other forms of caisson disease are due to the liberation of the air from the blood during too rapid decompression. That is the period of danger in caisson workers. Air bubbles are liberated in the joint fluids, cerebrospinal fluid, or spinal cord, and they may become paralyzed. Dr. Bassoe examined about a hundred and eighty men, over half of whom gave a history of trouble coming on after they came out of the caisson, particularly the cases of "blind staggers." They come out, are extremely dizzy, vomit, and often become deaf. It is of interest to figure out what happens in those cases, and whether it is liberation of air in the endolymph or a liberation of air in the auditory nerves—perhaps sometimes in the brain. There is here a possibility of the production of local lesions that might be of great interest to otologists. It has been suggested, for instance, that a local lesion might produce tone islands or other interesting disturbances, but so far as he knows trained otologists have rarely gone to work on these cases. Some work has been done in Vienna, where some very painstaking observations were made. It would seem that otology might profit from a study of this disease, because it is almost comparable to an experiment. You take a man from the normal pressure of fifteen pounds and put him in forty-five or fifty pounds, in so many minutes, and let him out in so many minutes, and then study the conditions present. We have this work going on nearly all the time, with wonderful opportunities for research. In the fatal cases we now know quite well what we are dealing with, namely, air bubbles in the blood. There is sometimes the condition spoken of as "chokes," in which the patient becomes suffocated and cyanotic. He sometimes recovers, but more often dies, and it has been found that the right auricle may contain huge air bubbles.

---

*See page 1121.

Dr. J. R. Fletcher thought that the cases referred to by Dr. Boot having the blind staggers are very likely serous labyrinthitis. It seems to him that where the air bubbles are liberated in the cochlea or in the semicircular canals, the damage would be very great to the organ. After mastoid operations we frequently see the production of serous labyrinthitis, and it acts in just the same manner that Dr. Boot mentioned in his paper, and it is not incomprehensible at all that the increase of pressure should produce a rather prolonged hyperemia, primarily, which would produce what we are justified in calling a serous labyrinthitis. In the cases in which the damage is very great there must be then a large quantity of fibrin in the serum, which becomes organized and does the real damage. So long as there is not much fibrin thrown out the recovery is probable, but sometimes very slow, even extending over years. In operative cases—not caisson workers—it may extend over a long time, but the majority recover.

Dr. Boot, in closing, said that the most characteristic thing in these cases is the degeneration of the auditory nerve, just as you see in deafnes resulting from occupation, boiler makers' deafness, for example—a slow degeneration that goes on, not at the time the men are working, but afterwards, after they have ceased working. This degeneration continues for a considerable length of time, and they lose the upper limit of hearing. That, he thinks, is the most characteristic lesion in these ear cases.

Another point of considerable importance is the fact that the lesion of the vestibular portion of the nerve, whatever it may be, seems to be recovered from completely. There was only one case where he did not get perfect response to the caloric test.

He doubts very much whether the points made by Dr. Wilson had very much to do with the case. When going under compressed air the endolymph would be forced out, and as they came out it would be forced back in again into the endolymph canals.

Dr. Wilson said that he thought the perilymph is the part that is first affected in the compression.

Dr. Boot thinks the blocking is purely middle ear trouble. the same condition we see in coming up and down in elevators, and which is relieved by swallowing, and these men are instructed to swallow. The other features he thinks are of the labyrinthine type—changes in the labyrinth.

He had hoped to be able to make some experiments with guinea pigs, in order to get the microscopic appearances of these ears, but as yet has not had an opportunity to do so.

Dr. Stein asked whether any of the cases had died and postmortems been performed, to which Dr. Boot replied that he knew of none who had died.

BOOK REVIEW.

Diseases of the Ear.

By PHILIP D. KERRISON, M. D. 588 pages, 331 illustrations in text, and two full pages in color. J. B. Lippincott Company, Philadelphia and London, 1913. $5.00.

The reviewer has read this book, the latest addition to otologic literature, with a great deal of interest, and regards it, on the whole, as one of the best of the textbooks on otology which the English language has yet produced. The style is pleasing, and the subjects are treated in a full and somewhat discussive fashion. While voicing the writer's own views, it is at the same time judicial and fair to the views of others, and fully recognizes that many procedures in otology are far from having reached finality. The author has the unfortunate habit of using the terms "author," "writer" and the personal pronoun "I" somewhat indiscriminately, and sometimes all three are on the same page; for instance, in two consecutive sentences on page 226 the terms "the writer" and "I know" are used. It seems to the reviewer that the person writing a book exponential of his own ideas should use the personal pronoun "I," and no other, or if adopting the term "author" or "writer" should stick to it.

The importance of the work justifies the consideration of some of its views a little in detail. A very good description is given of the anatomy of the ear at birth, and the changes which take place during the first two years, as well as of the topography of the drum. He denies the statement that the horizontal plate of the squama enters into the formation of the tympanic roof, and says that this is wholly formed by the superior plate of the os petrosum. The mastoid antrum is always present, even in fetal life, and is not a mastoid cell, but the posterior end of the tympanic vault.

The physiology of sound transmission is described not only clearly, but in such a way that it is read with genuine interest. A suggestion is given as to the discovery of malingering, namely, apply the caloric vestibular test whenever total deafness in one ear is claimed. If a quick and normal reaction is obtained, it is probable that the deafness is assumed. The treatment of furunculosis seems rational, and is given the attention which this common and distressing condition demands. The author regards furunculosis as requiring hospital attention, and finds that patients who are given absolute rest in bed do better than those who are treated as ambulatory cases. A general anesthetic is advised for any aural condition which may require very much manipulation, such as foreign bodies, incision of the drum, and the like. Ten per cent of scarlet fever cases develop tympanic disease. Measles give rise to many cases of acute otitis of severe type, often going on to mastoid trouble. Tinnitus aurium is considered at some length as to its causation, but unfortunately nothing of value is offered as to its treatment. Retraction of the drum is a sign of adenoids in children, and this has not as yet received sufficient attention. Classification of acute middle ear disease is given as acute catarrhal otitis media and acute purulent otitis media, followed by a very good account of the ordinary accepted pathology.

Catheterization is advised during the acute stage of otitis media after drum incision, and he does not believe that there is any danger of infection of the mastoid antrum from this source, as the line of least resistance is through the incised drum. Syringing rather than dry wicking is given the preference in the acute inflammations. In the case of antrum tenderness ice is allowed for twenty-four hours only. The indications as given for operating in acute mastoiditis are safely conservative, neither too hasty nor admitting of dangerous delay. Chronic middle ear suppuration is discussed in a very rational way, from all standponts, and from a personal viewpoint which appeals especially to the reviewer, who is inclined to treat many of his cases of chronic suppurative otitis media conservatively rather than radically. He states: "We should not be slaves to the dogma, too often repeated in otologic literature, that aural discharge is of itself necessarily a danger signal, or one which when persistent calls for operative intervention. If we can satisfy ourselves that the discharge is simply the logical product of exposed mucous membrane or healthy granulation tissue, and that there is no evidence of an active suppurative process involving the bone itself, we should not be too ready to interfere with nature's method of conservation." In certain forms of attic suppuration a simple mastoidectomy is advised, in order not to endanger the hearing power, the aditus being very carefully and thoroughly cleared of granulations and diseased bone.

In the section on otosclerosis are given three pictures from Professor Manasse's book, but there is no acknowledgment in the text nor any footnote telling definitely the source from which the illustrations are taken other than in the legend attached to the cuts, nor is there any reference in the text to Professor Manasse's theories. This is due to the fact that Dr. Kerrison's manuscript was in the hands of the printer when Professor Manasse's monograph was issued, and it was too late to make corrections in the text. The cuts were inserted with the permission of Professor Manasse's publishers, and in a later edition a full discussion of his theories will probably be given. On page 253, after epitomizing the symptoms, the remark is made that with such a picture the physician who fails to recognize otosclerosis with stapedial fixation is certainly not a practical otologist. As Professor Manasse has stated there is no symptom complex by which one can with certainty, during life, diagnosticate ostitis metaplastica or otosclerosis, such a statement seems hardly borne out by the facts. An absolutely hopeless prognosis, with reference to the value of any treatment in otosclerosis, is given. The anatomic point is made in the section of the labyrinth that the ordinary description of the scala tympani and scala vestibuli as being respectively the lower and upper cochlea channels is not only incorrect, but confusing, since with the head erect the central axis of the cochlea is not vertical but horizontal, and its direction from the vertical anteroposterior plane of the skull is horizontally forward and outward.

Much space is given to the labyrinth, and an endeavor made to make the whole subject as clear as possible. The rather new term in otologic literature, "arthrodial sense," is used as referring to the sense which tells the blindfolded individual of the position (in flexion or extension) of his joints and through this of his body generally. The reviewer has been unable to find this word, in the sense in which it is here used, in any of the dictionaries at his command, but understands that it is a rather common term in neurologic literature and discussion.

The symptomatology in the chapter on intracranial disease, and the relation of the ear thereto, is one of the most satisfactory sections, the valuation of each symptom in relation to possible lesions being most carefully considered. He advises in the section on infection of the sinus that the sinus be uncovered whenever there are any symptoms such as to suggest the possibility of sinus infection; that the sinus be exposed whenever any unusual amount of pus is encountered, in order to eliminate the possibility of an abscess in this location; also, whenever there are physical changes present which leave in doubt the question of the health of the sinus groove. No mention, however, is given as to uncovering the sinus in case it should be accidentally injured during the mastoid operation, but only directions as to caring for the hemorrhage. The reviewer is of the opinion that in case of accidental injury to the sinus it would be better to still further bare the sinus, enlarge the nick so as for the moment to produce free bleeding, and then to pack. He believes this is better than to leave a small opening which may, unknown to the operator, carry infection into the general blood stream, whereas, if the other treatment is given and the sinus packed off the risk from this source should be very slight.

In the chapter on brain abscess there is a careful review and study of the individual symptoms and their varying diagnostic value, followed by a most careful study of cerebellar abscess and the symptom relationship between it and cerebral abscess. He cites cases of injury to hearing from cutting of the stapes in paracentesis. Such a case has come within the knowledge of the reviewer.

The various steps of the simple mastoid operation are given in their proper order, and the reasons for each one individually stated. He does not advise removing any but the cortex of the tip, nor the removal of every vestige of diploeic or pneumatic cell; this being not only unnecessary, but physiologically impossible. Nor does he believe that a very smooth or even surface is essential for the formation of healthy granulations; on the contrary, to go further and curette the walls into surfaces of even and polished smoothness may reduce their vitality to a point seriously interfering with normal repair. The blood clot operation is described in the language of its originators, and is not favored. The point is made that in the radical operation the facial nerve is usually injured in its horizontal rather than its vertical portion, and with the curette rather than with the chisel. In delayed healing in the after-treatment of certain radicals which suppurate longer than is desired, he advises packing with boric acid, very nearly filling the canal, and then completing the filling with gauze, leaving it in for twenty-four hours, and replacing daily. Skin grafting is not advised at the time of the primary operation. He does not think one is ever justified in making definite promises to the individual as to the influence of the radical mastoid upon the hearing. In his own experience the hearing is oftener improved or unchanged rather than curtailed.

In the operative treatment of the labyrinth the various indications and the comparative advantages and disadvantages of the operations of Jansen, Hinsberg, J. D. Richards and the author's own method have been analyzed. Drainage in the great majority of cases is what the labyrinth operation should seek to accomplish. While hitherto an advocate of resection of the jugular vein, he is coming to the conclusion that simple ligation as practiced by the Boston school will answer. Most careful reasoning out of all the steps in all of the operative sections is given, especially that of the lateral sinus operation.

In exploring the dura in suspected brain abscess he thinks it an advantage to make parallel incisions at different points rather than to go diagonally in various directions from an initial point of entrance, as in the case of the latter all of the previous cuts may possibly become infected, whereas in the former method the cuts made which do not reach the abscess cannot be infected by the drainage from the abscess. An ingenious gauze wick for use in drainage is described and illustrated.

Adenoids are regarded as physiologic structures, a point of view which the reviewer has long held, and only when unduly enlarged to be considered as pathologic. Impaired hearing is one of the probable signs of their presence, and granulations along the posterior pharyngeal wall are pathognomonic. He remarks that the forcible use of the curette can remove not only lymphoid tissue, but also muscle tissue with resulting stenosis. Anyone who has examined with the finger postnasally some time after the curette operation for adenoids will be surprised to find how often adhesions of Rosenmüller's fossa to the posterior lip of the eustachian tube remain. He believes that if in the presence of acute otitis media adenoids are manifestly present, the operation for adenoids should be done at the same time as the drum incision. This can be done with one anesthesia, and many cases of acute ears will not clear up until the nasopharynx is cleared of the adenoid tissue. In considering the question of the relation of aural diseases to life insurance, while recognizing the possible gravity and risk of many lesions, he nevertheless believes that there are many cases in which, in spite of a large perforation allowing a certain amount of aural disease, the character of the lesion is such as to involve little or no risk to life. We have as yet no statistics which are at all reliable as showing the proportion of fatal cases of ear disease to the number of persons afflicted. Even those of the ear hospitals are not reliable, since large numbers of such cases are treated by private physicians, only severe cases coming to the ear hospital or specialist. The subject of artificial aids to hearing is rather slighted, as in most textbooks, a viewpoint which, to the reviewer, seems a mistake, as those artificial aids should be prescribed by the aurist in accordance with the needs of the individual, the same as glasses are prescribed by the oculist.

As showing the rapid advance in otologic science, we have chapters with such titles as "The Value of Salvarsan in Aural Disease," "The Use of Vaccines in Aural Disease," "Aural Disease in Life Insurance" and others, titles which we would have looked for in vain in most of the older otologic textbooks.

Examples of history charts, and the remedies and formulas which the author has found most useful, and a very good index complete the book. The illustrations are good, and add much to the value of the text.

Unfortunately the publisher has seen fit to print the book on paper about the weight of cardboard, making the volume very heavy to hold, and somewhat over four pounds in weight. It could easily have been printed on thinner paper, and would then have occupied not more than half or two-thirds its present bulk, and to the great comfort of the reader. It seems to be the policy of nearly every medical publisher to produce a volume of about the same size and price without much regard to the length of the manuscript. Attention should be drawn to this kind of padding oftener than it is.

INDEX OF AUTHORS.

ALEXANDER, G., 159, 885.
Andrews, A. H., 840.
Ard, 544.

BALLENGER, W. L., 836, 859, 862, 1187, 1190, 1193.
Ballin, M. J., 821.
Bassoe, Peter, 1194.
Beck, J. C., 255, 273, 558, 559, 578, 835, 842, 844, 849, 863, 913, 1166, 1168, 1180, 1188, 1192.
Beck, Oscar, 1099.
Berens, T. P., 234, 253, 433, 666.
Bergeron, J. Z., 578.
Birkett, H. S., 649, 826.
Blackwell, H., 538, 549.
Bliss, M. A., 869.
Boot, G. W., 265, 834, 841, 844, 1121, 1178, 1194, 1196.
Botella, Ernest, 883.
Bowen, H. M., 180.
Braislin, W. C., 1085.
Braun, A., 517.
Broeckaert, 890.
Brown, G. V. I., 271.
Brown, J. M., 1147.
Brown, R. H., 257, 258, 263.
Bryan, W. M., 811.
Bryant, W. S., 482, 540.
Butt, Wm. R, 201.

CALAMIDE, U., 895.
Canfield, R. B., 604.
Cavanaugh, J. A., 699, 839, 1174, 1180.
Chappell, W. C., 911.
Cheatle, A., 19.
Clark, J. Payson, 421.
Coakley, C. G., 909.
Cocks, G. H., 526.
Cohn, Felix, 527, 542, 831.
Combier, 895.
Corwin, A. M., 574.

DABNEY, VIRGINIUS, 889, 1057.
Dana, Chas. L., 239.
Danziger, E., 522, 525.
Davis, Geo. E., 519, 823.
Dean, L. W., 1077, 1166, 1167.
Dench, E. B., 229, 233, 244.
Denker, A., 1.
Dixon, Geo. A., 228, 369.
Duel, A. B., 234.

ELLEGOOD, J. A., 1067.
Emerson, Linn, 183, 333.

FLETCHER, J. R., 838, 851, 852, 853, 860, 865, 1195.
Fistre, 893.
Fowler, E., 523, 553, 557, 823.
Foy, Robert, 890.
Freedman, L. M., 186.
Freer, O. T., 558, 568, 848, 850.
French, Thos. R., 910.
Freudenthal, W., 464, 827.
Fridenberg, P., 250.
Friedberg, S. A., 835, 1176.
Friedenwald, Harry, 1090.
Friesner, I., 517, 522, 556, 820.

GAREL, 892.
Gelle, 880.
Glogau, O., 509, 525, 527, 825.
Graham, H. B., 798.
Greene, Jos. B., 782, 897.
Gruening, 526, 531.
Guisez, 904.

HALLOCK, W., 551, 556.
Hammond, P., 246, 652.
Harris, T. J., 475.
Haskin, W. H., 384, 525, 528.
Hastings, Hill, 1133.
Hawley, C. W., 851.
Hays, Harold, 235, 514, 525.
Held, R. J., 546.
Herzog, M., 1166, 1171.
Heymann, 896.
Hirsch, 828.
Holinger, J., 255, 257, 268, 837, 839, 843, 845, 847, 1173, 1186, 1187.
Holmes, E. M., 350.
Hopkins, F. E., 496.
Horn, H., 874, 989.
Hubby, L. M., 638, 820, 825, 831.

IGLAUER, S., 308.
Ingals, E. F., 563.
Ingersoll, J. M., 629.

JACKSON, CHEVALIER, 360, 889.
Jennings, J. E., 869.

KELSEY, A. L., 1147.
Kenefick, J. A., 527, 537.
Kenyon, Elmer L., 1110, 1187, 1189.
Kerrison, P. D., 239, 517, 548.
Kopetzky, S. J., 544.
Kosokabe, 879.
Kubo, 892.

Kulenkampff, 873.
Kyle, D. B., 912.

LAKE, R., 239, 254, 388.
Lamberson, Wm., 897.
Lammois, 881.
Law, F. M., 635, 826, 833.
Lederman, M. D., 431, 525, 547.
Leroux, 904.
Levy, Robt., 133.
Lewis, E. R., 264, 517.
Lewy, Alfred, 838, 1168, 1183, 1187, 1189.
Loeb, H. W., 871, 1177.
Long, Chas. H., 499, 558, 846.
Loughran, Robt. L., 886.
Luedde, W. H., 868.
Lynch, R. C., 1191.

MACKENZIE, GEO. W., 1020.
Mackenzie, John N., 1046.
Maclay, O., 256, 577, 623, 835.
Makuen, G. Hudson, 761.
Maloney, Wm. J., 531.
Manasse, 876.
Mauthner, 902.
Mayer, E., 901.
McCaw, Jas. F., 505.
McCoy, Geo. A., 822, 1073.
McKernon, J. B., 229, 232, 243.
Medan, S., 906.
Meierhof, 541.
Miller, H. E., 492, 867.
Moure, 892.
Myles, R. C., 807.

O'MALLEY, JOHN F., 1007.
Oppenheimer, S., 526, 547, 831, 890.
Ostrom, Louis, 560, 579.

PAGE, J. R., 321.
Phillips, Wendell, 517, 523, 826, 832.
Pierce, N. H., 834, 860, 863, 1081, 1167, 1168.
Pynchon, E., 258, 575, 578.

QUIMBY, J., 828.

RICHARDS, GEO. L., 672.
Richardson, C. W., 453, 488, 817.
Richez, 882.
Robinson, J. A., 228.
Roe, John O., 909.

SACHS, B., 521.
Sauer, W. E., 867, 872, 971.
Schlagel, 878.
Scripture, E. W., 536, 549.
Shambaugh, Geo. E., 259, 268, 303, 576, 659, 836, 839, 844, 849, 860, 862, 863, 1185.
Sheppard, Geo. A., 235.
Sluder, Greenfield, 725, 868, 870, 872, 907.
Smith, Harmon, 453, 769.
Smith, S. MacCuen, 769.
Sonnenschein, R. H., 256, 258, 579, 860, 1185.
Spencer, Selden P., 581, 889.
Stein, Otto J., 874, 1182.
Stewart, Wm. H., 677, 826, 832, 833.
Strouse, A. N., 522.
Stucky, J. A., 229, 236, 887.
Swain, Henry L., 1156.

TAYLOR, H., 550.
Theisen, C. F., 175.
Torrison, G. A., 255.
Tousey, S., 829.

VAIL, DERRICK, 572.
Van Wagenen, 832.
Veis, J., 879.
Voislawsky, A., 228, 832.

WALSH, JAS. J., 536.
Wilson, J. Gordon, 229, 265, 834, 843, 845, 846, 848, 864, 1179, 1196.
Winslow, J. R., 125.
Wishart, D. J. G., 453.
Wood, Geo. B., 894.
Wright, J. D., 532.

YATES, DAVID G., 886.

ZANGE, 875.
Zytowitsch, 877.

INDEX OF TITLES.

ABDUCENS paralysis associated with acute mastoiditis, Hays, 514, 525.
Abductor paresis, Stein, 1182.
Abscess and partial exfoliation of larynx cartilages from typhoid, Bryan, 811.
Abscess, brain, frontal lobe of otitic origin, Berens, 433.
Abscess, brain, otitic, Smith, 769; Sauer, 867.
Abscess, mastoid, Wilson, 848.
Abscess, pharynx, Swain, 1156.
Abscess, temporosphenoidal, Wilson, 848.
Abscess, temporosphenoidal, of otitic origin, McCaw, 505.
Accessory sinuses, Oppenheimer, 890.
Accessory sinuses, histologic pathology, Beck, 913.
Accessory sinuses, plaster casts, Loeb, 871.
Accessory sinuses, skiagraphs, Ballenger, 1190.
Adenoid vegetation, Fistre, 893.
Adenoids, removal, Beck, 273.
Adhesions, nasal, treatment, Maclay, 623.
After-nystagmus rotation, Fowler, 552.
Air embolus following puncture of maxillary sinus, Bowen, 180.
Anesthesia for operations in upright position, French, 910.
Anesthesia, local, for radical mastoid operation, Kulenkampff, 873.
Anesthesia, nerve trunk, and carbolization in nasal surgery, Sluder, 870.
Angina, Vincent's, Stein, 1182.
Antrum of Highmore, exploratory puncture followed by air embolus, Bowen, 180.
Aphthous ulcerations of upper air passages in pulmonary tuberculosis, Mackenzie, 1046.
Apparatus for controlling voice of singers and for teaching deaf to sing, Scripture, 549.
Arthritis deformans and otosclerosis, analogy, O'Malley, 1007.
Arthritis, rheumatic, cricoarytenoid, Calamide, 895.
Arytenoid perichondritis from typhoid, Bryan, 811.
Atresia external auditory canal, Lewy, 1168.
Atresia external auditory meatus, due to furunculosis, Long, 558.
Atresia, traumatic, auditory canal, with mastoidal complication, Danziger, 525.
Atrophic rhinitis, with ozena, Emerson, 333.
Auditory acuity enhanced by physiologic methods, Maloney, 531.
Auditory canal, external atresia, Lewy, 1168.
Auditory canal, external, filled by dilatation of jugular bulb, Lammois, 881.
Auditory canal, traumatic atresia, with mastoidal complication, Danziger, 525.
Auditory meatus, external, furunculosis causing atresia, Long, 558.
Auditory meatus stenosis, Long, 846.
Auditory nerve and salvarsan, Gelle, 880.
Aural diseases, ocular manifestations, Haskin, 384.

BACTERIA, passage through tonsillar tissue, Wood, 894.
Bacterium pneumoniae Friedlaender causing otitis media acuta, Zange, 875.
Bier's aspiration, results in otology, Botella, 883.
Bier passive hyperemia in nonpurulent affections of middle ear, Stein, 874.
Blood clot healing in mastoidectomy, Blackwell, 538.
Bone conduction in syphilis, Beck, 1099.
Brain abscess, otitic origin, Berens, 433.

Brain abscess, otitic, Smith, 769; Sauer, 867.
Bronchial calculus, history, Chappell, 911.
Broncholith, Chappell, 911.
Bronchoscopic removal of a collar button, Jackson, 360.
Bronchoscopy for multiple foreign bodies, Winslow, 125.

CAISSON workers' deafness, Boot, 1121, 1194.
Calculus, bronchial, Chappell, 911.
Cancer, larynx, treated by thyrotomy, Greene, 897.
Capsulated bacteria causing perforation petrous portion of temporal bone, Graham, 798.
Carbolization in nasal surgery, Sluder, 870.
Carcinoma epiglottis, primary, Mayer, 901.
Carcinoma superior maxilla, Beck, 559.
Cautery tip, V-shaped, Sluder, 872.
Cavernous sinus relation to sphenoid sinus, Sluder, 725, 907.
Cavernous sinus thrombosis, Horn, 989.
Cavum tympani, relation to ostium tympanum Zytowitsch, 877.
Cerebellar pontine angle tumor, Robinson, 228.
Cerebrospinal fluid, loss in certain conditions, Canfield, 604.
Cerebrospinal meningitis, Berens, 666.
Chicago Laryngological and Otological Society, 255, 558, 834, 846, 1166, 1182.
Cicatricial stricture of esophagus, Freer, 848.
Cicatricial stenosis of esophagus, Guisez, 904.
Cisterna magna drained in meningitis, Pierce, 1081, 1168.
Complications of submucous resection of nasal septum, Mackenzie, 1020.
Congenital osseous obstruction of postnasal orifices, Richardson, 488.
Congress, International Otological, in Boston, 1912, Holinger, 843.
Cranial nerves in relation with sphenoid, Sluder, 725, 907.
Cricoarytenoid arthritis, rheumatic, Calamide, 895.

DEAF instructed to sing by strobilion, Scripture, 549.
Deafness and vertigo as symptoms in tumor in cerebellar pontine angle, Robinson, 228.
Deafness, caisson workers', Booth, 1121, 1194.
Deafness from scarlet fever otitis and recognition of otitis interna serosa, Manasse, 876.
Deafness, progressive, Stucky, 229, 887.
Deafness, treatment by re-educating the hearing, Maurice, 211.
Decannulation and extubation after tracheotomy and intubation respectively, Jackson, 899.
Deflection, nasal, and nasal adhesions, Maclay, 623.
Deflection nasal septum, submucous operation complications, Mackenzie, 1020.
Deflections nasal septum, treatment by widening palatal arch, Brown, 271.
Digital evisceration of capsule following tonsil decapitation, Ellegood, 1067.
Diphtheria, Greene, 782.
Dysphagia in laryngeal tuberculosis, Combier, 895.

EAR connection with sexual apparatus, Dabney, 889.
Ear, internal, histology, Friesner and Braun, 517.
Ear, residual, Yates, 886.
Eighth nerve disease in connection with epidemic parotitis, Mauthner, 902.
Embolus air following puncture of maxillary sinus, Bowen, 180.
Embolus with sinus erosion, Schlagel, 878.
Epiglottis carcinoma, primary, Mayer, 901.
Esophageal bougie with spindle-shaped enlargement for dilatation of cicatricial stricture of esophagus, Freer, 848.
Esophagoscope for removal of false teeth from esophagus, Kyle, 912.

Esophagus, cicatricial stenosis, Guisez, 904.

Esophagus, cicatricial stricture, Freer, 848.

Esophagus, foreign bodies, Coakley, 909.

Esophagus, removal of foreign body from, Kyle, 912.

Essence of orange in anesthesia, French, 910.

Exenteration mastoid by nature, Smith, 769.

External auditory canal atresia, Lewy, 1168.

External auditory meatus, furunculosis causing atresia, Long, 558.

Extubation following intubation, Jackson, 899.

FIBROMYOMA, removal, hemophilia, Lederman, 431, 525.

Fifth cranial nerve, relation to sphenoid sinus, Sluder, 725, 907.

Fistula, double (right and left), right in horizontal semicircular canal, left in oval window, Fletcher, 851.

Foramen ovale, relation to ostium tympani, Zytowitsch, 877.

Foramen rotundum, relation to ostium tympanicum, Zytowitsch, 877.

Foreign bodies, bronchoscopy, Winslow, 125.

Foreign bodies in esophagus, Coakley, 909.

Fourth cranial nerve, relation to sphenoid sinus, Sluder, 725, 907.

Fracture of nose, Garel, 892.

Friedlaender's bacterium pneumoniae, causing otitis media acuta, Zange, 875.

Frontal mucocele, McCoy, 1073.

Frontal sinuses, plaster casts, Loeb, 871.

Frontal sinus, surgical, treatment, Myles, 807.

Furunculosis causing atresia of external auditory meatus, Long, 558.

GALVANIC nystagmus, Hubby, 638, 820.

Glaucoma, intraocular tension lowered by injection of sphenopalatine ganglion, Miller, 492, 867.

Graphite stains in membrana tympani, Voislawsky, 228.

HEARING organ malformation, congenital operation, Kosokabe, 879.

Hearing, reeducation, Richez, 882.

Hemophilia, removal of fibromyoma, Lederman, 431, 525.

Hemorrhage, nasal, following turbinectomy in a hemophiliac treated by injection of human blood serum, Emerson, 183.

Hodgkins' disease, tonsillar manifestations, Torrison, 255.

Hypophysis tumor, Sauer, 872, 971.

INFLAMMATION lateral columns of pharynx, Swain, 1156.

Internal ear histology, Friesner and Braun, 517.

Intracranial diseases, Beck, 1168.

Intraocular tension in glaucoma lowered by injection of the sphenopalatine ganglion, Miller, 492.

Intubation followed by extubation, Jackson, 899.

JUGULAR bulb, dilatation, filling middle ear and part of external auditory canal, Lammois, 881.

KERRISON'S diseases of the ear, 1197.

LABYRINTH anatomy, Friesner and Braun, 517.

Labyrinth, destruction of function following radical mastoid operation, Fletcher, 852.

Labyrinth diseases, Spencer, 581.

Labyrinth diseases, inflammatory, Phillips, 517.

Labyrinth, operation for cure of paracoustic vertigo and nystagmus, 321.

Labyrinth, operation for infection secondary to purulent otitis media, Shambaugh, 273.

Lactic ferment in rhinology, Medan, 906.

Laryngoscopy, suspension, Freudenthal, 464.

Larynx cancer treated by thyrotomy, Greene, 897.

Larynx tuberculosis, dysphagia, Combier, 895.
Larynx tuberculosis, early diagnosis, Lamberson, 897.
Larynx, tumor, Levy, 133.
Larynx, ulceration from typhoid, Bryan, 811.
Lateral columns pharynx inflammation, Swain, 1156.
Lateral sinus thrombosis. Friedenwald. 1090.
Lung stone, history, Chappell, 911.

MALFORMATION, congenital, of hearing organ. plastic operation, Kosokabe, 879.
Malignant tumors, nasopharynx, Kelsey and Brown, 1147.
Mastoid, abscess, and temporosphenoidal abscess, Wilson, 848.
Mastoid bone development, illustrated by radiographs, Stewart, 677, 826.
Mastoid diagnosis aided by radiography, Law, 635, 826.
Mastoid exenteration by nature, Smith, 769.
Mastoid operation and vaccines, Braislin, 1085.
Mastoid operation, clinical indications, Bryant, 482.
Mastoid operation, laboratory and X-ray indications, Dixon, 369.
Mastoid operation, radical, Hammond, 652.
Mastoid operation, radical, essentials for proper healing of wound cavity, Loughran, 886.
Mastoid operation, radical, followed by destruction of function of labyrinth, Fletcher, 852.
Mastoid operation, radical, under local anesthesia, Kulenkampff, 873.
Mastoid radiography symposium, Birkett, Stewart, Law, 826.
Mastoid skiagraphs, Ballenger, 1190.
Mastoidal complication in traumatic atresia of auditory canal, Danziger, 525.
Mastoidectomy, blood clot healing, Blackwell, 538.
Mastoiditis, acute, with paralysis of abducens nerve, Hays. 514, 525.
Mastoiditis, operative findings, Ingersoll, 629.
Mastoiditis, pathology, illustrated with radiograms, Birkett, 649, 826.
Maxilla, superior, carcinoma, Beck, 559.
Maxilla, superior, odontoma, Dean, 1077.
Maxillary sinuses. plaster casts, Loeb, 871.
Maxillary sinus, puncture followed by air embolus, Bowen, 180.
Meatus, auditory, stenosis, Long, 846.
Meckel's ganglion, intranasal treatment, Holmes, 350.
Membrana tympani, graphite stains, Voislawsky, 228.
Meniere's symptom complex, Hastings, 1133.
Meningitis, cerebrospinal, Berens, 666.
Meningitis with drainage of cisterna magna, Pierce, 1081, 1168.
Meningoencephalitis, acute otogenous, Alexander, 885.
Menthol poisoning, Leroux, 904.
Mica splints for treatment of nasal synechia, Moure, 892.
Middle ear filled by dilatation of jugular bulb, Lammois, 881.
Middle ear, gross lesions, Wilson, 848.
Middle ear, nonpurulent affections treated with Bier's passive hyperemia, Stein, 874.
Middle ear suppuration, Alexander, 159.
Middle ear tuberculosis, primary, Long, 499.
Monochord, Struycken-Schaefer, 1183.
Mucocele, frontal, McCoy, 1073.
Mucoperichondrium, relation to periosteum of nasal septum, Maclay, 835.

NASAL accessory cavities, diseases, Oppenheimer, 890.
Nasal adhesions, treatment, Maclay, 623.
Nasal cavities, tuberculosis, Maclay, 256.
Nasal diseases. ocular manifestations, Haskin, 384.

Nasal deflection and treatment of nasal adhesions, Maclay, 623.
Nasal necrosis, treatment with radium. Broeckaert, 890.
Nasal septum deflections treated by widening palatal arch, Brown, 271.
Nasal septum, periosteum, relation to mucoperichondrium, Maclay, 835.
Nasal septum, submucous resection, Butt, 201
Nasal synechia, treatment by means of mica splints, Moure, 892.
Nasal tissues, intranasal transplantation, Iglauer, 308.
Nasal tuberculosis, Freer, 558.
Nasal voice. Kenyon, 1110. 1187.
Nasopharynx tumor, malignant, Kelsey and Brown, 1147.
Necrosis, nasal treatment with radium, Broeckaert, 890.
Nerve trunk anesthesia and carbolization in nasal surgery, Sluder, 870.
New York Academy of Medicine, 228. 239. 517. 525, 538, 820, 826
Nitrous oxid gas, essence of orange, ether and sequestration in general anesthesia, French, 910.
Nose connection with sexual apparatus. Dabney, 889.
Nose fractures, Garel, 892.
Nose, pneumococcus infection, Theisen, 175.
Nystagmus and paracoustic vertigo cured by labyrinth operation. Page, 321.
Nystagmus, galvanic, Hubby, 638, 820.
Nystagmus, vestibular, Flechter, 853.

OCULAR manifestations in nasal and aural diseases, Haskin, 384.
Odontoma superior maxilla. Dean. 1077, 1166, 1175.
Ostium tympanicum of tubae eustachii, relation to fundus cavum tympani. foramen rotundum and foramen oval, Zytowitsch. 877.
Otitic brain abscess. Berens, 433; Smith. 769; Sauer. 867.
Otitic temporosphenoidal abscess. McCaw, 505.
Otitis interna serosa, Manasse, 876.
Otitis media acuta due to bacterium pneumoniae Friedlaender, progress and treatment, Zange, 875.
Otitis media, chronic suppurative—treatment, Richards, 672.
Otitis due to scarlet fever resulting in sudden deafness, Manasse, 875.
Otogenous meningoencephalitis, acute, Alexander, 885.
Otolaryngology, teaching, Wishart, Smith, Richardson, 453.
Otology and aspiration of Bier, Botella, 883.
Otology from medical point of view, Spencer, 889.
Otosclerosis, Holinger, 843, 847.
Otosclerosis and arthritis deformans, analogy, O'Malley, 1007.
Ozena, treatment by functional nasal reeducation, Foy, 890.
Ozena with atrophic rhinitis, Emerson, 333.

PALATAL arch; widening for correction of deflections of nasal septum, Brown, 271.
Paracoustic vertigo and nystagmus cured by labyrinth operation. Page, 321.
Paresis abductor. Stein, 1182.
Paralysis abducens associated with acute mastoiditis, Hays, 514, 525.
Parotitis epidemic in connection with diseases of eighth nerve, Mauthner. 902.
Perforation petrous portion temporal bone due to capsulated bacteria, Graham, 798.
Perforation Shrapnell's membrane, Holinger, 255.
Periosteal flap necrosis followed by sinus thrombosis. Glogau, 509, 525.
Peritonsillar phlegmon, Hopkins. 496.
Pharynx abscess, Swain, 1156.
Pharynx, lateral columns inflammation. Swain. 1156.
Phlegmon. peritonsillar, Hopkins, 496.
Phlegmon upper respiratory tract. Roe. 909.
Physiology of semicircular canals. Shambaugh, 259.

Pin-swallowing, Friedberg, 835.
Plastic operation for congenital malformation of hearing organ, Kosokabe, 879.
Pneumococcus infections of the nose and throat, Theisen, 175.
Poisoning from use of menthol, Leroux, 904.
Polyp, sphenochoanal. Kubo, 892.
Postnasal orifices, congenital osseous obstruction, Richardson, 488.
Pyemia with sinus erosion, Schlagel, 878.

RADIOGRAMS illustrating pathology of mastoiditis, Birkett, 649, 826.
Radiographic findings illustrating anatomic development of mastoid bone, Stewart, 677, 826.
Radiography as aid in mastoid diagnosis, Law, 635, 826.
Radiography mastoid, symposium, Birkett, Stewart, Law, 826.
Radium as treatment for nasal necrosis, Broeckaert, 890.
Reeducation of hearing in treatment for deafness, Maurice, 211.
Resonators, with special reference to Schaefer apparatus, Sonnenschein, 256.
Residual ear, Yates, 886.
Rheumatic cricoarytenoid arthritis, Calamide, 895.
Rhinitis, atrophic, with ozena, Emerson, 333.
Rhinolaryngology practice, bearing of nasal voice, Kenyon, 1110, 1187.
Rotation after-nystagmus, Fowler, 553.

ST. LOUIS Medical Society, otolaryngological section, 271, 870, 872.
Salvarsan and acoustic nerve, Gelle, 880.
Sarcoma of tonsil or tonsillar manifestation of Hodgkins' disease, Torrison, 255.
Schaefer apparatus, resonators, Sonnenschein, 256.
Semicircular canals, physiology, Shambaugh, 259.
Semicircular canal, horizontal, fistula, Fletcher, 851.
Septum deflection, submucous operation complications, Mackenzie, 1020.
Septum, nasal, deflections treated by widening palatal arch, Brown, 271.
Septum, nasal, periosteum relation to mucoperichondrium, Maclay, 835.
Septum, nasal, submucous resection, Butt, 201; Ostrom, 560.
Sequelae of radical tonsil removal, Dabney, 1057.
Sequestration in anesthesia, French, 910.
Sexual apparatus connection with ear, nose and throat, Dabney, 889.
Shrapnell's membrane, perforation, Holinger, 255.
Sinus, accessory, skiagraphs, Ballenger, 1190.
Sinus thrombosis following necrosis of periosteal flap, Glogau, 509, 525.
Sinus, maxillary, puncture followed air embolus, Bowen, 180.
Sinus, lateral, thrombosis, Friedenwald, 1090.
Sinus, frontal, surgical treatment, Myles, 807.
Sinus erosion with embolus and pyemia, Schlagel, 878.
Sinus, cavernous, thrombosis, Horn, 989.
Sinuses, accessory, histologic pathology, Beck, 913.
Sinuses, accessory, plaster casts, Loeb, 871.
Sixth cranial nerve, relation to sphenoid sinus, Sluder, 725, 907.
Skiagraphs, mastoid, Ballenger, 1190.
Skiagraphs nasal accessory sinuses, Ballenger, 1190.
Specialist training, Harris, 475.
Speech, conversational, in relation to whispered voice, Veis, 879.
Sphenochoanal polyps, Kubo, 892.
Sphenoid sinuses in plaster casts, Loeb, 871.
Sphenoid sinus relations cavernous sinus and third, fourth, fifth, sixth and Vidan nerves, Sluder, 725, 907.
Sphenopalatine ganglion injection for lowering of intraocular tension in glaucoma, Miller, 492.

Stammering, Makuen, 776.
Stenosis auditory meatus, Long, 846.
Stenosis, cicatricial, esophagus, Guisez, 904.
Streptothrix organism causing cavernous sinus thrombosis, Horn, 989.
Stricture esophagus, cicatricial, Freer, 848.
Strobilion, an apparatus for controlling voice of singers and for teaching the deaf to sing, Scripture, 549.
Submucous resection, Butt, 201.
Submucous resection operation, complications, Mackenzie, 1020.
Superior maxilla carcinoma, Beck, 559.
Superior maxilla odontoma, Dean, 1077.
Synechia, nasal, treatment by mica splints, Moure, 892.
Syphilis and bone conduction, Beck, 1099.

TEMPORAL bones, dissected, Haskin, 528.
Temporal bone, petrous portion, perforation due to capsulated bacteria, Graham, 798.
Temporal bone section, Friesner and Braun, 517.
Temporal bone, symmetry, Cheatle, 19.
Temporosphenoidal abscess, Wilson, 848.
Temporosphenoidal abscess of otitic origin, McCaw, 505.
Third cranial nerve relation to sphenoidal sinus, Sluder, 725, 907.
Throat, connection with sexual apparatus, Dabney, 889.
Throat, pneumococcus infection, Theisen, 175.
Thrombosis, sinus, cavernous, Horn, 989.
Thrombosis, sinus, following necrosis of periosteal flap, Glogau, 509, 525.
Thrombosis lateral sinus, Friedenwald, 1099.
Thyrotomy for cancer of larynx, Greene, 897.
Tonsil decapitation followed by digital evisceration of its capsule, Ellegood, 1067.
Tonsil, focus for systemic infection, Shambaugh, 659.
Tonsil function, Freedman, 186.
Tonsil removal sequelae, Dabney, 1057.
Tonsil sarcoma, Torrison, 255.
Tonsillar manifestation of Hodgkins' disease, Torrison, 255.
Tonsillar tissue, report concerning passage of bacteria through, Wood, 894.
Tonsillectome, Jennings, 869.
Tonsillectomy, results after operation, Clark, 421.
Topography tympanic cavity, Cavanaugh, 839.
Tracheotomy followed by decannulation, Jackson, 899.
Transplantation, intranasal, of nasal tissues, Iglauer, 308.
Tuberculosis, larynx, dysphagia, Combier, 895.
Tuberculosis, larynx, early diagnosis, Lamberson, 897.
Tuberculosis middle ear, primary, Long, 499.
Tuberculosis, nasal, Freer, 558.
Tuberculosis nasal cavities, Maclay, 256.
Tuberculosis, pulmonary, and aphthous ulcerations, Mackenzie, 1046.
Tumor cerebellar pontine angle, Robinson, 228.
Tumor hypophysis, Sauer, 872, 971.
Tumor larynx, Levy, 133.
Tumor nasopharynx, malignant, Kelsey and Brown, 1147.
Turbinectomy in a hemophiliac, hemorrhage treated by injection of human blood serum, Emerson, 183.
Tympanic cavity topography, Cavanaugh, 699, 839.
Typhoid fever causing ulceration of larynx, perichondritis of arytenoid cartilages, abscess and partial exfoliation of both cartilages, Bryan, 811.

ULCERATION, aphthous, of upper air passages in pulmonary tuberculosis, Mackenzie, 1046.
Upper air passages, aphthous ulceration in pulmonary tuberculosis, Mackenzie, 1046.
Upper air tract, vasomotor disturbances, Richardson, 817.
Upper respiratory tract phlegmon, Roe, 909.

VACCINES after mastoid operation, Braislin, 1085.
Vasomotor disturbances, upper air tract, Richardson, 817.
Vertigo, Lake, 239.
Vertigo and deafness as symptoms of tumor in cerebellar pontine angle, Robinson, 228.
Vertigo, clinical and therapeutic study, Lake, 888.
Vertigo, paracoustic, and nystagmus cured by operation on labyrinth, Page, 321.
Vestibular apparatus, model reconstruction, Lewis, 517.
Vidian nerve relation to sphenoid sinus, Sluder, 725, 907.
Vincent's angina, Stein, 1182.
Vocal cords, unilateral affection, Heymann, 896.
Voice, nasal, Kenyon, 1110, 1187.
Voice, whispered, in relation to conversational speech, Veis, 879.

X-RAY and laboratory indications for mastoid operation, Dixon, 369.

(19)
746-4

RF
1
A64
v. 22

Annals of otology, rhinology
and laryngology

1913

Biological
& Medical
Serials

PLEASE DO NOT REMOVE
CARDS OR SLIPS FROM THIS POCKET

UNIVERSITY OF TORONTO LIBRARY

STORAGE